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Endowment fund of the International Association for Dental Research (as of Dec. 1, 1944): $1,370.98. There have been no expenditures from this fund.

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Journal of Dental Research: The Association's official publication. See register of officers and editors on page 2 of cover.

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

The Twenty-third General Meeting will be held at the Hotel Continental, Kansas City, Mo., March 17 and 18, 1946, and coordinated with the meetings of the American Association of Dental Schools and Omicron Kappa Upsilon to be held on March 19–21.

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THE DESTRUCTIVE EFFECT OF CITRATE VS. LACTATE IONS ON RATS' MOLAR TOOTH SURFACES, IN VIVO[1]

F. J. McClure, Ph.D. and Stanley J. Ruzicka, D.D.S.

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The enamel and dentin of rats' molar teeth were observed in previous studies by McClure (1) to be notably destroyed when dilute acid solutions and certain acid drinks and beverages replaced drinking water in rats' diets. These observations have been confirmed and extended by Restarski, Gortner and McCay (2). While the destructive effects of the several drinks and beverages studied (1) were due largely to their acidity, the question arose, as to whether any one specific organic ion or ions might not have been particularly active in destroying the tooth surfaces. Because of its presence in citrus drinks and for reasons related to certain chemical properties which will be discussed later, the citrate ion seemed especially interesting for study in these regards, and it appeared of considerable interest to compare the effect of citrate with lactate because of the prevailing association of lactic acid with dental caries etiology. A special requirement of the experiment was to eliminate, or at least to greatly reduce, the possibility of any oral surface effects due to acidity. The experiment was planned therefore to observe any action of pure dilute solutions of sodium lactate and sodium citrate on rats' tooth surfaces in vivo, under practically neutral conditions such as prevail within the oral cavity.

PLAN OF EXPERIMENT

The general plan of the studies is indicated in Table I. Rats were compared by trios, each trio being composed of litter mates of the same sex. The acidity of lactate and citrate drinking fluids was comparable to the pH of human saliva, the pH of the drinking fluids being respectively in 3 separate studies as follows: pH 5.5–5.6, pH 6.5–6.7, and pH 7.0–7.2. In experiments A and C (Table I) the fluid intake of rats receiving lactate and citrate fluids was equalized. The rats' drinking habits as observed in Experiments A and C did not indicate preference for one test fluid over another; therefore, even though fluid intakes were not equalized, in Experiment B there is reason to believe that the average total quantities of test-fluids ingested did not vary greatly. The diet, consumed ad libitum, was made up as follows: corn starch 66%, whole milk powder 27%, yeast 5.0%, cod liver oil 1.0%, sodium chloride 1.0%.

After terminating the periods of exposure to the drinking fluids (see Table I) the rats were sacrificed, and the heads were autoclaved to facilitate removal of the

[1] Received for publication January 12, 1946.

soft tissues. The molar teeth from the right side were dissected out, dried at 110° for 1 hour and weighed (Table I). In experiments B and C the clinical crowns of the molar teeth were scored for relative acid effects, according to the system proposed by Restarski, Gortner and McCay (3), Table II. The lingual surfaces of typical teeth were photographed and both transverse and longitudinal sections were prepared for photomicrographs (*fig. 2*) in order to illustrate the relative decalcifying effects of the citrate and lactate ions.

TABLE I

Weight of molar teeth of rats exposed to water, lactate and citrate drinking fluids

EXPERIMENT	A			B			C		
Drinking fluid	water	lactate	citrate	water	lactate	citrate	water	lactate	citrate
Concentration of drinking fluid (%)		2.2	1.7		2.6	2.0		2.2	1.7
Normality of drinking fluid		0.20	0.20		0.25	0.25		0.20	0.20
pH of drinking fluid	7.0	5.5–5.6	5.5–5.6	7.0	6.5–6.7	6.5–6.7	7.0	7.0–7.2	7.0–7.2
Number of rats averaged	11	11	11	12	12	12	12	12	12
Days on experiment (days)	120	121	119	136	136	136	142	151	150
Total fluid drunk (ml.)	1865	1925	1970				2128	2309	2306
Final body weight (gm.)	156	158	150	170	145	150	205	215	219
Average weight of molar teeth	Right molars			Right molars			Left molars		
Upper 1st molar (mg.)	15.0	15.0	14.2	15.9	16.0	15.3	16.7	16.6	16.7
Upper 2nd molar (mg.)	9.3	9.2	8.7	10.1	10.1	9.7	10.6	10.6	10.2
Upper 3rd molar (mg.)	4.5	4.4	4.2	5.3	5.2	5.1	5.5	5.3	5.8
Upper molars (mg.)	28.8	28.6	27.1	31.2	31.3	30.2	32.8	32.5	32.7
Standard deviation	2.68	1.88	2.13	1.63	1.92	1.79	1.80	1.89	1.72
Lower 1st molar (mg.)	14.2	14.4	12.6	15.2	15.2	14.5	15.9	15.8	15.7
Lower 2nd molar (mg.)	9.8	9.8	8.6	10.9	10.7	10.0	11.2	11.2	11.1
Lower 3rd molar (mg.)	5.5	5.4	4.6	6.1	6.1	5.6	6.2	6.1	5.9
Lower molars (mg.)	29.5	29.6	25.8	32.2	32.0	30.2	33.3	33.1	32.7
Standard deviation	2.35	2.01	2.08	1.76	1.45	1.42	1.35	1.87	1.45
Upper and lower molars (mg.)	58.3	58.2	52.9	63.4	63.3	60.3	66.1	65.6	65.4
Standard deviation	4.95	3.81	3.83	3.50	3.06	3.16	2.55	3.58	3.05

RESULTS OF EXPERIMENT

The action of the citrate drinking fluid at pH 5.5–5.6 on the rats' exposed tooth surfaces resulted in statistically significant differences in weights of molar teeth of control rats vs. rats drinking the citrate solution (Exp. A, Table I). The average losses in weight (difference in the weight of molar teeth of citric acid rats vs. control rats) were as follows: Comparing upper right molar teeth the difference was 1.7 ± .66 mg.; for lower right molar teeth 3.7 ± .64 mg.; and for all upper and lower right molar teeth the difference in weight was 5.4 ± .89 mg. Under the same conditions the lactate solution caused no loss in weight of right molar teeth.

In Experiment B the drinking fluids had a concentration of .25N and a pH of 6.5–6.7. At this pH also, the loss in weight observed in molar teeth of rats drinking the citrate fluid was statistically significant as compared with the teeth of control rats (Table I). Differences in weights of molar teeth of citric acid rats

TABLE II

Numerical evaluation of destructive action of citrate ion vs. lactate ion on rats' molar teeth—average score for individual teeth

[According to arbitrary grading system proposed by Restarski, J. S., Gortner, R. A., Jr., and McCay, C.M. (Ref. 3)]

Experiment	B			C		
Number of rats (trios)	15	15	15	12	12	12
Drinking fluid	water	lactate	citrate	water	lactate	citrate
pH of drinking fluid	7.0	6.5–6.7	6.5–6.7	7.0	7.0–7.2	7.0–7.2
Upper right 1st molars	0.1	0.5	4.5	0.0	0.3	1.9
Upper right 2nd molars	0.1	0.5	4.5	0.0	0.3	2.2
Upper right 3rd molars	0.1	0.3	3.4	0.0	0.2	1.6
Average upper right molars	0.1	0.4	4.1	0.0	0.3	1.9
Upper left 1st molars	0.1	0.5	3.3			
Upper left 2nd molars	0.1	0.5	3.7			
Upper left 3rd molars	0.0	0.3	2.9			
Average upper left molars	0.1	0.4	3.3			
Average upper molars	0.1	0.4	3.7	0.0	0.3	1.9
Lower right 1st molars	0.3	1.6	5.7	0.0	0.8	2.8
Lower right 2nd molars	0.3	1.6	5.5	0.0	0.8	2.6
Lower right 3rd molars	0.1	1.1	5.3	0.0	0.5	1.7
Average lower right molars	0.2	1.4	5.5	0.0	0.7	2.4
Lower left 1st molars	0.1	1.2	5.3			
Lower left 2nd molars	0.1	1.2	5.6			
Lower left 3rd molars	0.1	0.7	5.2			
Average lower left molars	0.1	1.1	5.4			
Average lower molars	0.1	1.3	5.5	0.0	0.7	2.4
Average for molar teeth	0.1	0.9	4.6	0.0	0.5	2.1

vs. controls (Exp. B) were as follows: Comparing upper right molar teeth the difference was 1.0 ± .69 mg.; for lower right molar teeth 2.0 ± .64 mg.; and for all upper and lower right molar teeth the difference in weight was 3.1 ± 1.34 mg. As in Experiment A, the lactate solution caused no loss in weight of molar teeth that could be detected by the methods followed.

In Experiment C, no significant losses were observed in weight of the molar teeth which had been subjected to either the citric or the lactic solutions.[2]

As was pointed out in a previous study (1), differences in weight of molar teeth tend to minimize the corrosive action of drinking fluids because a large portion of the weight of a tooth consists of the unexposed root. Likewise, only the lingual surfaces have any appreciable contact with drinking fluids and during the drinking process the lower molars are more in contact with drinking fluids than are the upper molars. Therefore, the surface effects of these drinking fluids were studied further by means of the arbitrary scoring system proposed by Restarski, Gortner and McCay (3), *fig. 1*. This system was made use of in Experiments B and C, the results of which are recorded in Table II.[3] Photographs of representative teeth and longitudinal and transverse sections associated with the different numerical scores are shown in *fig. 2*.[4]

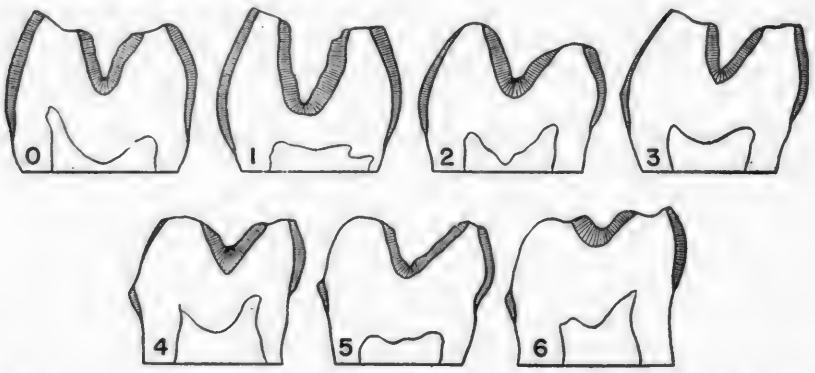

Fig. 1. Tracings from photomicrographs of longitudinal ground-sections of typical mandibular first molars of rat, showing various stages of enamel destruction. Numbers 0 to 6 indicate scores assigned to teeth, tooth score increasing with progressive loss of enamel from exposed lingual surfaces. [After Restarski, Gortner, and McCay, (3)]

It should be noted regarding the data in Table II that average tooth scores appear for control (water) rats' teeth in Experiment B and for both groups of lactate rats (Exp. B and C). These scores are attributed to individual abnormalities and artefacts on tooth surfaces not strictly related to any destructive effect of the drinking fluids consumed by the rats. There is obviously no expectation that distilled water would affect tooth surfaces. As regards the average scores for the "lactate" teeth we are inclined to minimize their significance,

[2] There is reason to believe that a significant weight difference might have developed in the teeth of these citrate rats, had they been continued on experiment for a longer period of time.

[3] We wish to express our indebtedness to Dr. Restarski, Dr. Gortner and Dr. McCay for advising us personally in the scoring of these tooth surfaces.

[4] We are greatly indebted to Passed Assistant Surgeon Kenneth M. Endicott, of the Division of Pathology, National Institute of Health, for assistance and advice in obtaining these photographs.

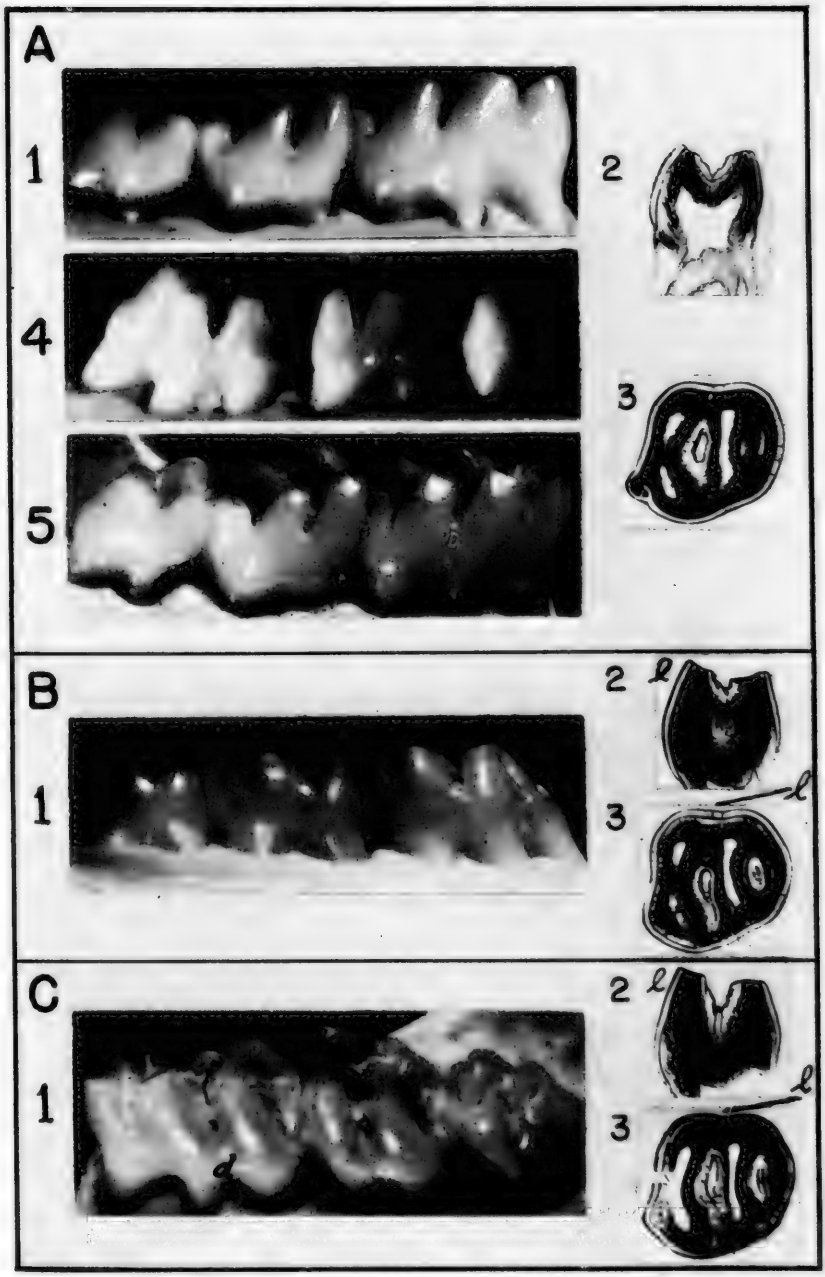

Fig. 2. Photographs of lingual surfaces of rat molars accompanied by photomicrographs of ground-sections, illustrating various stages of enamel destruction produced by citrate and lactate solutions (pH 6.5–6.7). Tooth scores indicate degree of destruction and accompanying descriptions define criteria on which scoring was based. (See p. 7 for detailed legend).

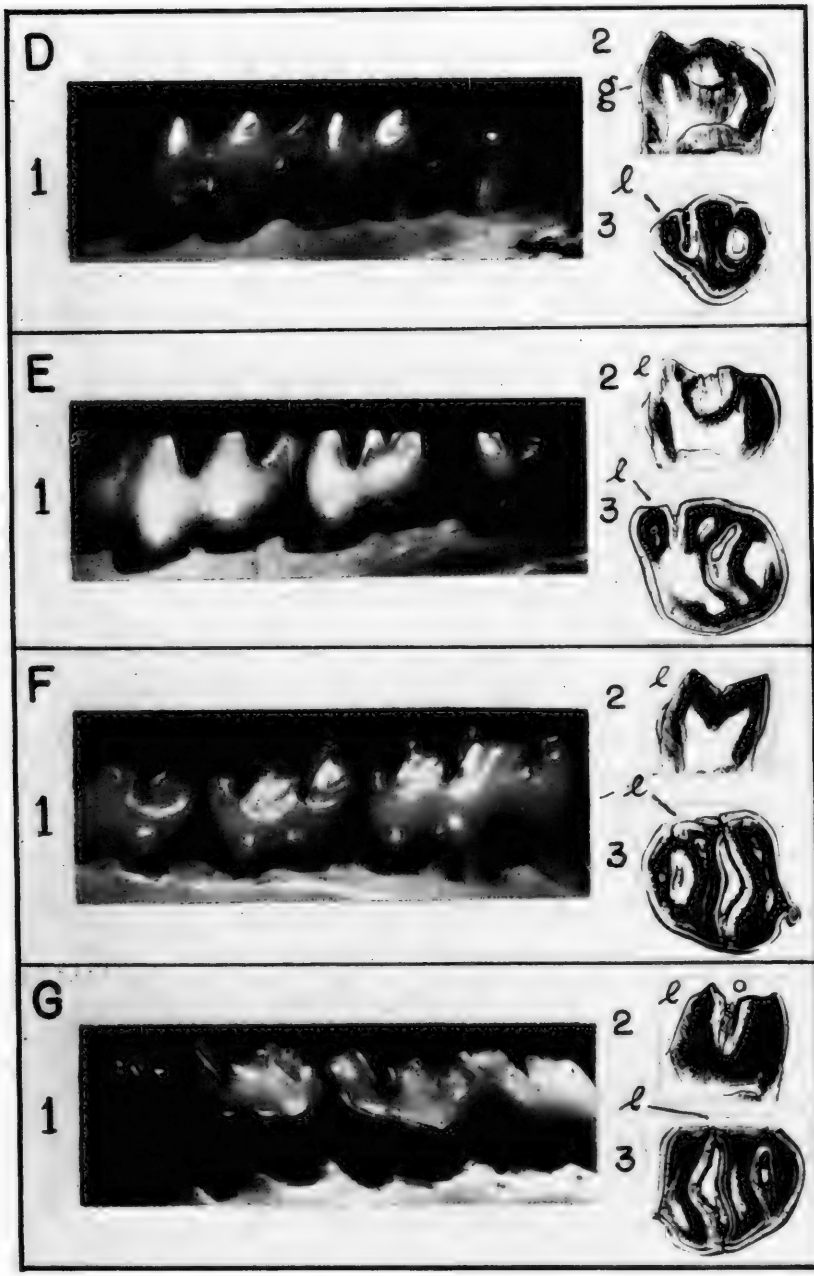

Fig. 2. Photographs of lingual surfaces of rat molars accompanied by photomicrographs of ground-sections, illustrating various stages of enamel destruction produced by citrate and lactate solutions (pH 6.5–6.7). Tooth scores indicate degree of destruction and accompanying descriptions define criteria on which scoring was based. (See pp. 7 and 8).

although it is not to be denied there is a suggestion of a very mild effect of this ion on tooth surfaces (see *fig.* 2-c, 1). The extreme effect of the citrate ion over

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A. Tooth Score 0.
(1) Lower molars. Control rat.
(2) Longitudinal section through central cusp of lower first molar. Control rat.
(3) Transverse section through gingival third of lower first molar. Control rat.
(4) Upper molars. Control rat.
(5) Lower molars. Lactate rat. Scutiform abrasion on linguo-occlusal surfaces of lingual cusps in control rats and lactate rats where destructive action was not great enough to alter lingual surfaces at the tips of the cusps. Believed an artefact due to occlusal wear and not considered in evaluating tooth scores.

B. Tooth Score 1.
(1) Upper molars. Citrate rat. First indication of enamel destruction: lingual enamel highly polished; flattening of lingual surfaces. Note loss of contour of central cusp of second molar.
(2) Longitudinal section through central cusp of lower first molar. Lactate rat. Lingual enamel flattened (*l*); lacks full contour as in (A, 2).
(3) Transverse section through gingival third of lower first molar. Lactate rat. No change in thickness of lingual enamel (*l*) as effect is more apparent above gingival margin.

C. Tooth Score 2.
(1) Lower molars. Lactate rat. Slight etching of lingual enamel beginning at gingival margin of clinical crown produces line of demarcation (*d*). Slight rounding of cusps at occlusal edge.
(2) Longitudinal section through central cusp of lower second molar. Lactate rat. Slight etching has thinned lingual enamel (*l*) appreciably enough to produce tapering toward occlusal, thus giving cusp rounded appearance.
(3) Transverse section through gingival third of lower first molar. Lactate rat. No appreciable lingual enamel destruction (*l*), although gross specimen (C, 1) shows line of demarcation (*d*) beginning at gingival margin of clinical crown.

D. Tooth Score 3.
(1) Upper molars. Citrate rat. Beginning of slight ridge formation at gingival margin of clinical crown. Mild destruction of lingual enamel.
(2) Longitudinal section through central cusp of upper first molar. Citrate rat. Ridge starting at linguo-gingival margin (*g*) with enamel thinning and tapering toward occlusal.
(3) Transverse section through gingival third of upper third molar. Citrate rat. Mild destruction of lingual enamel (*l*). Lowest tooth score at which effect in gingival third area is observed histologically.

E. Tooth Score 4.
(1) Upper molars. Citrate rat. Moderate destruction of lingual enamel with some exposure of dentin producing definite ridge. "Island" of exposed dentin in center of affected cusps.
(2) Longitudinal section through central cusp of upper second molar. Citrate rat. Moderate destruction of lingual enamel (*l*). Well defined ridge. Exposure of dentin first noted. Enamel tip at occlusal encloses "island" of dentin.
(3) Transverse section through gingival third of upper second molar. Citrate rat. Moderate loss of lingual enamel (*l*).

F. Tooth Score 5.
(1) Lower molars. Citrate rat. Severe destruction of lingual enamel with exposure of dentin from gingival margin of clinical crown to occlusal surface. Marked ridge at gingival margin.

and above the lactate ion is none the less apparent in the numerical scoring data for Exps. B and C.

The effect of the pH of the drinking fluids is apparent in the 2 sets of scoring data, the citrate and possibly the lactate solutions being less reactive at pH 7.0–7.2 (Exp. C) than at pH 6.5–6.7 (Exp. B). The citrate solutions, according to the weight data shown in Table I, were likewise more destructive at pH 5.5–5.6 (Exp. A) than at pH 6.5–6.7 (Exp. B) or at pH 7.0–7.2 (Exp. C). In general this relation of effect to acidity was to be expected since previous studies in vivo (1, 2) and certain results obtained with dentin and enamel in vitro (4, 5, 6) have pointed to the sensitivity of dental tissues to even mildly acid solutions.

Further evidence of the action of the citrate ion, particularly on the rats' tooth surfaces, is presented in the actual photographs of tooth surfaces of representative rats and also in the transverse and longitudinal sections of representative teeth as shown in *fig. 2*. There is conclusive evidence therefore in the weight data shown in Table I, in the scoring data presented in Table II, and in the photographs shown in *fig. 2*, together with the accompanying descriptive information, that the citrate ion had a seriously destructive action on these rats' dental tissues. The action occurred at a pH of 5.5 through the range of pH 7.0–7.2. No comparable effect of the lactate ion is apparent in the data.

DISCUSSION

The most interesting result of this experiment is the pronounced action of the citrate ion on the exposed surface of rats' teeth. As was indicated above, the selection of the citrate ion for study was suggested not only by its presence in citrus drinks, but also by the purely chemical data pertaining to the precipitation and solution of sparingly soluble salts, $CaCO_3$, and $Ca_3(PO_4)_2$ (7, 8, 9, 10) and particularly in results showing the solubilizing action of the citrate ion on insoluble calcium salts (11, 12, 13, 14). Greenwald (11) succeeded in demonstrating remarkable changes produced by a number of organic ions on the ap-

(2) Longitudinal section through mesial cusp of lower second molar. Citrate rat. Marked ridge at gingival margin of clinical crown with severe destruction of lingual enamel (*l*) and exposure of dentin from ridge to occlusal surface.

(3) Transverse section through gingival third of lower first molar. Citrate rat. Lingual enamel destroyed (*l*). Lowest tooth score at which dentin is exposed in gingival third area.

G. Tooth Score 6.

(1) Lower molars. Citrate rat. Almost complete destruction of lingual enamel of clinical crown to the degree where even the enamel separating the cusps has been obliterated to form an almost continuous ridge, mesial to distal, at the gingival margin. Marked exposure of dentin with some destruction as evidenced by crateriform depressions on lingual surfaces and notching of cusps.

(2) Longitudinal section through central cusp of lower first molar. Citrate rat. Definite ridge. Complete destruction of lingual enamel (*l*) of clinical crown. Exposure of dentin with some destruction on lingual and occlusal surface (*o*).

(3) Transverse section through gingival third of lower first molar. Citrate rat. Complete lingual enamel destruction (*l*). Dentin exposed with some destruction.

parent solubility products of $CaSO_4$, $Ca_3(PO_4)_2$ and $CaCO_3$. He emphasized the physiological significance of his results as follows:

"It is quite evident from these experiments that the presence of only a small amount of an organic anion tremendously changed the apparent solubility products of calcium sulfate, carbonate and phosphate. It is obvious that the precipitation or solution of calcium phospate, either in vitro, or in vivo, is not entirely a matter of calcium phosphate and hydrogen ion concentration but may be due to a change in concentration of some other ion." (11).

Of special significance in these regards are the results of experiments designed to study calcification in vitro. Shipley, Kramer and Howland (10) thus observed no abnormal effects of chloride, acetate or lactate ions on the calcification process but "when calcium was present as citrate no calcification was obtained" (10). Shear and Kramer (12) and later Shear, Kramer and Resnikoff (13) in extensive conductivity titration studies found that calcium chloride when titrated with sodium citrate gave abnormal results as compared with titrations of sodium chloride with sodium citrate. This abnormal reaction involving calcium and citrate ions is regarded as "direct evidence for the binding of calcium ions by sodium citrate in some kind of soluble complex" (12). Calcium ions are removed from solution presumably by the formation of a soluble, although slightly ionized, calcium citrate complex, the nature of which remains unknown.

With regard to the citrate-calcium effect in particular, Lansing and Scott (15) have reported an interesting perfusion experiment with cats using a 0.9% sodium citrate solution. Control and citrated perfusion samples of liver, kidney, duodenum, rectus abdominus muscle, and femoral nerve were studied by means of micro-incineration and electron microscopy. The results show that calcium and magnesium were removed from the nucleus, cytoplasm, and cell membrane of cells perfused with sodium citrate. This result suggests reactions involving calcium and citrate ions to form soluble complexes similar perhaps to those occurring in the chemical studies cited above.

Numerous in vitro studies relative to solubility of dentin and enamel in various acids appear in the literature (4, 5, 6). In general, these studies incriminate neither the lactate nor citrate ions as having unusual solubility effects on dentin and enamel. The suggestion was made, although the experimental evidence is quite limited, that enamel solubility may be affected by different acid-anions (5).

In our observations on the destructive effect of the citrate ion on the dentin and enamel of rats' teeth there is now available additional evidence of the solubilizing effect of the citrate ion on an insoluble calcium phosphate complex. The action seems to be an interesting example of what Shear, Kramer and Resnikoff (13) observed in vitro, i.e., "the binding of calcium ions by citrate ions in some kind of soluble slightly ionized complex." It is noteworthy that with our results this action possibly may now be extended to include a vital calcified body tissue, in situ.

In relation to the etiology of dental caries this result may be of some interest; first, because it appears to represent a non-acid decalcification of dentin and enamel in situ, and second, because the effects of the lactate ion are insignificant when compared with the citrate ion. The results indicate beyond doubt that

the citrate ion is much more destructive to dental tissues under neutral conditions than is the lactate ion. However, at this time evidence does not permit identifying the citrate ion with the etiology of dental caries since the gross appearance of the attacked tooth surfaces bears no resemblance to dental caries.

On the other hand this action of citrate on dental tissues may be viewed as a purely chemical phenomena in which case it seems justified to emphasize the non-acid characteristics of the reaction, and to suggest that non-acid decalcification may have an important rôle in the etiology of dental caries. Although W. D. Miller's (16) well known theories of acid decalcification in relation to dental caries etiology are widely accepted, very little evidence has been presented to explain how acid decalcification of dental tissues occurs in the presence of practically neutral saliva.[5] The citrate's solubilizing effect observed in this study occurred under practically neutral conditions such as are found in the normal oral cavity. Our results seem to emphasize that a non-acid decalcification of dental tissue may be brought about by the metabolic products of bacteria which are active in the mouth.[6] These results illustrate that a factor other than acidity may be present which modifies or even produces a decalcification of dentin and enamel in situ.

It should now be pointed out that while the appearance of the destroyed surfaces of these rats' teeth did not resemble ordinary dental caries, there is a similarity between this phenomenon and erosion in human teeth. While erosion is as much an enigma as is dental caries, it may be suggested, in the light of this result, that saliva (or localized fluid exudates bathing tooth surfaces) may contain citrate or other anions which affect calcium solubility and advance the destruction of tooth surfaces locally.

Finally it may be noted that there is the suggestion in these and previous results (1) that excessive consumption of foods and beverages containing the citrate ion might have harmful local effects on human dental tissues. It is a matter of pure speculation, however, as to what relation foods and beverages which contain free citrate ions bear to the initiation or prognosis of dental caries.

Investigations are planned of other anions whose properties suggest actions similar or more pronounced than citrate on tooth surfaces.

[5] Failing to explain how acid decalcification of dental tissues is compatable with the pH of saliva, there has been a new approach in studies of intra-oral hydrogen ion concentration (17, 18) and of the pH of carious tooth material and especially of the nature and acidity of incubation products of dental plaque material (17, 18, 19, 20, 21, 22, 23, 24).

[6] Stephan (18) has discussed the possibilities of agents other than acids which may "effect decalcification of teeth in caries." He cites the possibility of removal of phosphate ions by phosphorylation and especially the possibility,—as has received substantial support from the results of our citrate ion studies—"of removal of calcium ions by complex ion formation with polycarboxylic acids" (18).

Incubation studies of dental plaque material suggest to Muntz (24) "The rapid production of acids other than lactic and volatile acids may be of some significance." Certain of these acids according to Muntz may be of the dicarboxylic type, "such as succinic or malic," which could form undissociated soluble complexes with calcium. Formation of volatile acids in plaque material was demonstrated to occur at neutrality in vitro.

SUMMARY

A study was made of the effect of citrate and lactate anions on rats' tooth surfaces, in vivo. Citrate and lactate drinking fluids adjusted to pH 5.5–7.2 were consumed in place of drinking water by young growing rats, and under these practically neutral conditions the citrate ion had a marked local decalcifying action on lingual tooth surfaces. No such action was caused by the lactate ion.

The action of citrate is explained as due to the binding of the calcium of the dentin and enamel by citrate to form a soluble calcium citrate complex. This in vivo action by citrate was predicted in certain fundamental studies of the chemistry of calcium and citrate ions.

The destruction of dental tissue by citrate, in itself, bears no gross similarity to ordinary dental caries. The process is believed to be a non-acid decalcification brought about by a specific organic anion, i.e., citrate, under practically neutral conditions. The suggestion is made that in considering the etiology of dental caries less emphasis be placed on acid-decalcification, and that the possibilities of a non-acid decalcification brought about by bacterial metabolites, be given more attention in dental caries research.

The destructive action of citrate on dentin and enamel surfaces suggests certain similarities with erosion observed in human teeth.

The results suggest further studies in vivo regarding surface effects of other anions in addition to citrate, as well as effects of certain foods and beverages on tooth surfaces.

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USE OF PENICILLIN AS A ROOT CANAL TREATMENT[1]

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The use of penicillin in the treatment of root canal infections has already been reported by Weiner (1). Large doses were given (5,000 to 30,000 units) by infiltration at the root apex with novocain, and also by syringing the root canal. No mention is made of any treatment left in the canal between visits. The number of cases was too small for statistical conclusions but a great deal of promise was evidenced.

Adams (2) reported the use of penicillin in root canal therapy by the method of injecting into the canal with a suitable 27 gauge needle. A total of 12 cases is included in the report. The author, himself, stated a difficulty in evaluating the method: "We have no way of knowing how much of the solution flows back out of the canal after the infected area is filled." Considering the dense nature of bone and the incompressibility of fluids, it is very likely that the "injection" of 2 cc. of penicillin containing 5,000 units, really constitutes an irrigation of the canal, rather than an injection into tissue.

To help standardize the utilization of this drug in the field of root therapy several important points must be elucidated. These are as follows: 1. How sensitive are the root canal organisms, in vitro, to the new drug? 2. What dosage can safely be employed and how may it be conveniently administered? 3. What is the relative freedom from irritation effects in this particular tissue, the facial bone? 4. How long does the extremely labile penicillin remain active when sealed in the root canal?

A clinical and laboratory study was made to answer these questions. A series of patients were accepted for treatment, most of them with moderate or well advanced abscesses or granulomas. Standard methods were employed in treatment, including the use of the rubber dam, rigid asepsis, mechanical measurement as a guide in instrumentation and culturing of root canal fluid using Difco brain heart infusion broth tubed in $\frac{1}{2}''$ by $3''$ tubes (4). These details have been well described by several writers (4 and 5). Cultures were made from all canals, and examinations from these made with the Gram stain and by the hanging drop method.

EXPERIMENTAL

A total of 36 cases are included, chiefly anterior teeth. Of these, 5 were diagnosed as pulpitis with slight or no radiographic change. The balance (31 cases) consisted of varying stages of pulp necrosis with small to large radiographic

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changes. Subdivisions might be made into abscess or granuloma, but these are neither certain nor fruitful.

Penicillin sensitivity of root canal organisms. The prevalent root canal organism is accepted as being the *Streptococcus viridans.* It has been reported (3) that while these bacteria are more resistant than other cocci, they are susceptible in varying degree to penicillin, even in its early impure form. It was thought worth while to routinely verify the sensitivity of root canal organisms, *qualitatively.* Growth from all cases was plated on brain heart agar, in $2\frac{1}{2}''$ Petri dishes. On these dishes were placed small (5 mm.) blotting paper discs, which had previously been wetted with 5 to 50 unit of sodium penicillin. The discs were prepared in large numbers at one time, dried immediately in a simple KOH drier and kept dry in the icebox. They were found to be unchanged in potency for at least 4 months. When placed on an inoculated Petri dish they will produce a

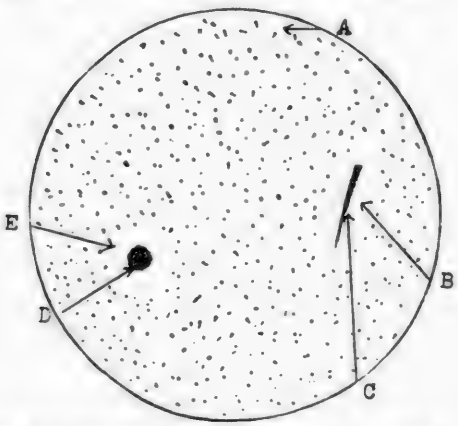

Fig. 1. Agar Plate Bacteriostasis Test. A. Bacterial colonies, B. Clear inhibition zone, C. Absorbent point with Ca penicillin in oil, D. Test disc, 5 oxford units, E. Clear inhibition zone.

large or smaller circle of clear bacterial inhibition. From all 36 cases tested, the organisms of 33 were found to be penicillin sensitive. The organisms from 2 cases turned out to be *Monilia albicans*, and from 1 was *B. coli.*[2] These 3 cases were resistant to penicillin.

Penicillin Dosage. Considering the great instability of the moistened drug, coupled with its freedom from toxicity, the dose should be as large as possible. The value of a mere wash in the root canal is highly dubious because of the small contact time and the negligible penetration into the tissues which are the sites of the infection. A solution, even when considered strong from other standards, would leave only a few units in the root canal if it were sealed in the root canal as a dressing. The small amount of penicillin soon disappears either through diffusion, oxidation or hydrolysis. Large doses may nevertheless be utilized as root canal medication. One way is to prepare absorbent points, cut to suitable length, moistened with thick drops of penicillin made on sterile glass slide, and then desiccated as already described and kept cold. Such medicated points made up

[2] Identifications by Dr. Henry Bartels, Columbia University Dental School.

in large numbers at one time, will not deteriorate and may be treated to carry 1000 to 2000 units of dry penicillin. They are convenient to use, and when used as a pumping wash into the apical tissue, with a drop or so of sterile water, the strength of this wash is of the order 20,000 or more units per cc.

In later cases a suspension of calcium penicillin[3] in oil was employed, 100,000, units per cc. This may be carried into the canal with a small glass syringe with a 25 gauge needle of suitable length. It is estimated that 5,000 units may easily be sealed in the average anterior root canal.

Irritation Effects. In the 36 cases reported in most of which penicillin was used many times, no irritation effects such as periapical periodontitis or odontalgia were observed. Injections of 10,000 to 15,000 unit doses placed in Cook ampules of cobefrin-2% novocain were given into the periapical regions of 3 volunteers. Anaesthesia was good and recovery usual. No pain or other untoward incidents appeared.

The Ca penicillin oil suspension was tested by injecting a few drops subcutaneously in the forearm of 1 person. There was a painless fibrous reaction, the slight swelling being slowly resorbed (2 months time), leaving a slight thickening of the skin and brown pigmentation. At no time was there pain or signs of tissue necrosis.

Stability of Penicillin Sealed in the Canal. It was soon found that even large doses of sodium penicillin disappeared when sealed in the canal with an open foramen. Dressings containing 1,000 units originally, exhibited only slight bacteriostatic powers when removed 24 hours after having been placed. They revealed no inhibitory effect on inoculated Petri dishes. This may be due to rapid diffusion, oxidation, hydrolysis or enzyme action. It indicates the need for a more stable method of using penicillin in the root canal.

Ca penicillin in oil (Upjohn) seems to fill this need admirably. Dressings removed up to 1 week after being placed, still exhibited distinct bacteriostatic power when plated on Petri dish as described (*fig. 1*).

CONCLUSIONS

A study of the use of penicillin as a medication within the root canal in 36 cases shows that most organisms found in the canal are penicillin sensitive. Sodium penicillin, and calcium penicillin in oil (Upjohn) are nonirritating. Sodium penicillin, even in large doses, disappears from the canal in 24 hours. Ca penicillin in oil maintains its bacteriostatic effects for upwards of 1 week when sealed in the root canal as a treatment.

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[3] Supplied for experimental purpose by Upjohn and Co.

THE EDITOR'S VIEWPOINT

The Editor wishes to experiment with a page of comments. If the readers indicate a desire for it to be continued such a policy will be discussed with our Board of Editors. Until such a time, we can call this an editorial research problem.

Will the atomic energy studies and the atomic bomb have any effect on dentistry? This may appear to be a foolish question. The atomic bomb has stimulated an interest in science so that many youngsters will be attracted by the glamour. From this group many will fall by the wayside, but some of our great scientists of the future will be from the glamour-attracted youths. Dental science should benefit from this wave of enthusiasm. We believe that dental students are showing a quickened interest in the sciences. Let us hope that the development of a weapon of destruction will aid mankind in this way.

The paper by F. J. McClure and S. J. Ruzicka, beginning on page one of this issue, presents a delightfully refreshing facet in the yet unfinished gem we hope to cut from our crude basic material. To us, the possibilities of applying this work on the citrate ion to the problem of erosion are most intriguing. We shall look forward to the results of salivary citrate analyses from erosion-free and erosion-positive individuals. We hope to see such a study instituted in our own clinics and elsewhere. We are not overlooking the possibility of applying this ion study to other problems.

The international flavor of the JOURNAL is returning. In this issue, we publish a paper from Kenya (p. 17). Aside from the dental aspects of Dr. Schwartz' contribution, we thoroughly enjoyed the background of geography and ethnics. Since Dr. Schwartz is a dental surgeon we hope that he will compile his data in greater detail in the future. We would like to know what teeth decayed and what method of examination was used.

Drs. Weisberger and Johnson (p. 35) are carrying forward the study of nutritional requirements of *L. acidophilus*. Gradually, a great amount of material on this organism is being accumulated. Not only does dental research benefit, but also bacteriology and other sciences gain from such basic studies.

We knew that penicillin would pop up in one of our manuscripts sooner or later. The scarcity of such material in a dental research journal and abundance in clinical periodicals may suggest that much of the use of this drug is based on empiricism. The paper by Maurice Buchbinder (p. 13) evaluates penicillin in root canal therapy.

Dr. J. P. Lazansky (p. 21) presents a study of the effect of "Septochem" as a dental caries' retarder. Light of this character should be cast on all new methods and drugs. We are pleased that the author used the commercial product as the cry of "special value" may be raised by commercial interests if laboratory compounded substitutes are tried. Clinical "impressions" cannot be substituted for objective data.

Dr. Gottlieb has two interesting contributions (pp. 25 and 29) on odontoblastic functions and on dentin resorption and deposition. The ideas here presented afford food for thought. That electric current experiment (pp. 33–34) appears worthy of more extensive investigation.*

H. V. Meredith has collected and analysed much material on eruption of deciduous teeth (p. 43). We would enjoy more correlations and compilations done in this accurate and thorough manner. Isn't it remarkable that only $\frac{1}{4}$th of the studies of deciduous tooth eruption appeared in dental journals. Why?

The printer, Editor and Business Manager have agreed to meet all deadlines "on the line" in 1946. Our printing schedule has been changed to 45 days (a saving of a full month) and we expect every future issue to appear on time. Papers should be in the readers hands in 60 to 90 days after receipt, if they are received in good form. Contributors should be certain that their manuscripts are presented as they are intended to be interpreted and in good grammatical style. They should have their references in the form suggested on the page facing page one and their illustrations neatly prepared. Line drawings and graphs should be on white paper in black ink. When in doubt, consult a scientific or medical "style book." Confidentially, we rewrote three of the papers in this issue, partly rewrote another, and sent two back to their authors for revision. We do not intend to do that year in and year out (we've had ten years of it now), because we know our authors aren't making their best efforts. When you receive proof, mail it back to the Editor (not to the printer), within 48 hours. Delay on your part, may delay an issue of the JOURNAL.

H. B. G. R.

*We have just noted that the electric stimulus study was published by Gottlieb and Orban. in Z. f. Stom. 28: 1208, 1930.

THE TEETH OF THE MASAI[1]

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The author has examined the teeth of the Masai tribe in Kenya in an effort to contribute to the metabolic and inner secretory theories of the etiology of periodontal disease and to the question of natural immunity in dental caries.

The Masai are a Nilotic people, related to the Nandi and Lumbwa of East Africa. Practically nothing is known about the early history of the Masai but it is assumed that they originally inhabited the land between the north of Lake Rudolf and the Nile. From there they migrated in a south-easterly direction. In about 1900, when the administration of East Africa was instituted and settlement began, the Masai were inhabiting the area on both sides of the Uganda Railway from Molo to Naivasha and in the South from Nairobi to Kiu. Because of increase of white settlers in the Protectorate, the Masai were moved to 2 reserves in 1904. The southern reserve was on both sides of the Rift Valley from Nairobi to the Tanganyika boundary. The northern reserve, known as the "Laikipia" reserve, was between the Uaso Nyiro and Lake Baringo. In 1913 the southern reserve was extended and the Laikipia Masai moved south. The reserve now is 288 miles long and 94 miles wide, an area of 14,600 square miles (9,000,000 acres). The reservation is divided into 2 administrative parts, the western part administered from Narok and the eastern part from Kajiado. This discussion deals only with inhabitants of the eastern region.

The Masai are a pastoral people. The males of the tribe are divided into boys, warriors and elders. The boys become warriors, called *Morans*, after circumcision which is usually between the ages of 13 and 17. The warriors do no work; they stroll about the bush and live in a state of free love with immature girls, a tradition remaining from the days of tribal warfare. The Moran rarely lives with his family but lives with all the other Morans in large locations miles from their original homes. After a periof of 5 to 10 years, he settles down, marries and starts the normal life of an elder. This is to herd cattle and look after the children.

The Masai live in small villages, called *maniattas*, of as many as 30 huts. These are built of mud covered with cow-dung. The building of the huts is the women's duty. The huts are arranged in a circle or square around a central yard into which the cattle are driven for the night. This yard is covered with layers of old cow-dung, and is swarming with millions of flies. Inside the hut a fire burns and there is no ventilation, smoke visibly irritating the eyes of the in-

[1] Received for publication November 15, 1945.

[2] Proof read by Dr. Barbara Goldberger, Queens Village, L.I., N. Y.

TABLE I

AGE	I CALCULUS				II. MARGINAL GINGIVITIS		III. PERIODONTOCLASIA				IV. CARIES			V. MISSING TEETH				VI. REMARKS	TOTAL
											Number of			Front		Back			
	a	b	c	%	No.	%	1	%	2	%	Cases	Teeth	%	Cases	Teeth	Cases	Teeth		
Children Under 14	4				2		2				1	6						1 open bite, 1 malocclusion, 1 syphilis	33
Men Under 20	15	2			13		3							1	1	1	2	1 open bite, 4 malocclusion, 1 supern. tooth	46
Women Under 20	1	2			1														10
	24			26.96	16	17.97	5	5.6			1	6	1.12	1	1	1	2	8 anomalies	89
Men Under 30	32	4			24		8				2	4		5	8	8	10	8 malocclusion, 1 retained dec. tooth	81
Women Under 30	13	2			7		4		1		3	9		2	5	4	12	5 malocclusion	43
Men Under 40	28	7	1		22		9		5		2	4		11	15	7	12	5 malocclusion, 1 retained dec. tooth	62
Women Under 40	6	3			5		6				1			3	5	1	1	2 malocclusion	28
	79	16	1																
	96			44.86	58	27.1	27	12.61	6	2.8	8	17	3.7	21	33	20	35	22 anomalies	214
Men Over 40	28	25	15		47		17		4		3	9		19	52	43	150	5 malocclusions	83
Women Over 40	9	8	1		18		11				3	6		6	15	10	42	2 malocclusions	22
	37	33	16																
	86			81.9	65	61.9	28	26.66	4	3.8	6	15	5.71	25	67	53	192	7 anomalies	105

habitants. Outside, flies sit in clusters on the faces, and even on the eyes, of the people while they wander through the foot-deep mud from one hut to another. In these surroundings, Masai are born, raised and live their lives, generation after generation.

The principal food of the Massai is milk, meat and blood, the latter obtained by bleeding their cattle. The protein consumption, therefore, is extremely high, while that of carbohydrate is negligible. The Masai have ample means with which to get maize meal and fresh vegetables but these foodstuffs are known only to the few who have worked in town. It is impossible to induce the Masai to plant their own maize or vegetables near their huts. They excuse themselves because they must move to other places with their cattle in case of dry weather.

This diet, disbalanced to the protein side, was the author's reason for selecting the Masai for study. It is seldom that one finds a people living under such unchanging diet and with poor hygienic conditions of centuries' duration. These conservative, independent and listless people are the richest and most backward tribe of East Africa. Owing to unhygienic conditions and tribal customs, they are ravaged by disease, especially venereal diseases, and are dying out. However, the Masai men are recognized as having good physiques and handsome features.

DATA

Collecting data was difficult due to their small scattered maniatta and independence. With the aid of Mr. Windley, the District Officer, and Dr. Horowitz, the Masai physician, data were accumulated from 408 Masai (Table I).

Age groups. The given age groups are used because the native does not know his exact age and the social structure is of little value. One's own judgement corroborated by the native clerk is the best guide.

Periodontium. The three groups for salivary calculus (a. very little, b. some, c. much) are purely clinical. For periodontoclasia there are 2 groups, 1. teeth with pockets of less than $\frac{1}{10}$ inch each, well fixed with no sign of looseness and 2. teeth with deep pockets, loose and with possible paradontal abscess.

Condition of Teeth. The two lower central teeth, which are extracted in childhood, are not counted in the recorded data, nor are the third molars.

Intermaxillary relations, anomalies and general health conditions are noted in Column 6 of Table I.

DISCUSSION

The only prior report on the teeth of the Masai known to the author is that of Dr. C. Philips in his Medical Survey made in 1930–31. Philips' report gives a different picture which the present author considers to be due to the fact that Dr. Philips was not a dentist and therefore unacquainted with the clinical aspects of periodontal disease. Moreover, he may have mistaken discolored enamel for caries. He gave the occurrence of "alveolar pyorrhea" as 59–69% for adults, 16–30% for Morans, and 8–18% for children. Table I shows comparable figures for marginal gingivitis, but not for periodontoclasia. For caries, he gave the

following data: 1.4–7.0% for adults, 0.0 for Morans, 0.25–1.0% for children. There does not appear to be any basis for evaluating or comparing these figures.

Study of the first 3 columns of Table I shows a rather geometric progression in age groups and almost direct proportions between calculus and gingivitis or periodontoclasia. This indicates that the author's observation is correct, i.e., that periodontal disease in the Masai is overwhelmingly "Schmutz pyorrhoea." Columns 4 and 5 show progressive frequency of caries and loss of teeth with age. The low caries-frequency and low number of lost teeth are extraordinary. Between the ages of 20 and 40, 214 individuals lost only 68 teeth altogether. The few anomalies recorded in Column 6 (Table I), further testify to the excellent condition of the teeth of the Masai. Although this is true, nearly all in the higher age group (92.36%) have diseased gingivae.

CONCLUSIONS

Caries is negligible among the Masai, only 38 cavities being observed in 408 individuals. This rate (0.09 cavities per mouth) is the lowest reported in all races examined by scientists.

Marginal gingivitis and salivary calculus, have high rates as a result of lack of oral hygiene.

The relative caries immunity of the Masai, appears to the author, to be due to their protein diet. I assume this to be rich in vitamins and mineral salts. Their teeth have hard use due to chewing coarse food in the form of tough meat.

All questions involved deserve scientific investigation.

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THE EFFECT OF TOPICAL "SEPTOCHEM" APPLICATIONS ON EXPERIMENTAL CARIES[1]

J. P. LAZANSKY[2]

From the University of Rochester, School of Medicine and Dentistry, Division of Dental Research

The apparent ability of topical applications of fluoride to inhibit dental caries suggests that other compounds might bring about a similar effect. Recently, considerable publicity has been given to the claim that topical applications of "Septochem" (1, 2) will inhibit dental caries in humans and a limited amount of clinical evidence has been advanced to support this belief. According to the available information "Septochem" is a preparation having as its chief components silver nitrate and indigisol. Frequent claims have been made for the caries inhibitory ability of one of these (silver nitrate). The distributor's directions indicate that "The treatment is based on the fact that dental caries is an invasion by micro-organisms of the organic matter of the tooth, especially the prism sheaths and the lamellae of the enamel. Impregnation of these organic roads with a strong antiseptic solution is supposed to make the tooth resistant to dental caries by rendering the organic roads impassable to micro-organisms." Since the preparation has been made available to the profession for caries prophylaxis it seemed advisable to investigate the caries inhibiting properties under controlled conditions such as those afforded by animal experiments.

Previous to 1942 the albino rat was the animal of choice for studies of caries inhibition. However, recently Arnold (3) indicated the apparent suitability of the Syrian hamster for investigations of this type. This finding has been elaborated upon by others (4, 5) and it now seems probable that the hamster will supplant the white rat in most experimental caries studies. Accordingly, in our study of the influence of topical applications of "Septochem" on experimental caries, we have included hamsters as well as rats.

EXPERIMENTAL

Three comparable groups of hamsters and rats were selected for study. Group I hamsters were placed on the diet of Arnold (3) at 40 days and served as a control. The other groups received the same diet. However, Group II was given 2 full mouth and Group III 2 half mouth (left side) topical applications of "Septochem" on the 5th and 19th day of the experiment. Group I rats were placed on the Hoppert, Webber, Canniff (6) diet at 40 days of age and served as a control.

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The other groups were maintained on the same diet but in addition were subjected to topical "Septochem" applications on the 5th and 19th days of the experiment. Group II had full mouth and Group III half mouth (left side) applications. It is of interest to note that experimental diets differ considerably in their physical properties, the hamster diet (Arnold) being finely ground and the rat (H.W.C.) diet containing coarse particles.

TABLE I

Caries activity in Syrian hamsters and rats

	SYRIAN HAMSTERS			RATS		
	Group I	Group II	Group III	Group I	Group II	Group III
	Diet and treatment					
	Fine corn, H_2O Control	Fine corn, H_2O, Septochem Full mouth	Fine corn, H_2O, Septochem Left side	Coarse corn, H_2O Control	Coarse corn, H_2O, Septochem Full mouth	Coarse corn, H_2O, Septochem Left side
No. of animals	10	6	6	14	7	10
Caries-free animals	none	none	none	none	none	none
Molar teeth examined	120	72	72	168	84	120
No. of carious molars	77(64%)	55(65%)	46(76%)	42(25%)	40(48%)	49(41%)
Maxillary	44	30	24	27	17	28
Mandibular	33	25	21	15	23	21
No. of cavities						
Maxilla	64	64	43	38	22	34
Mandible	48	51	47	22	36	38
Right						
Maxilla	38	32	18	19	8	14
Mandible	27	32	23	7	17	17
Left						
Maxilla	26	32	25	19	14	20
Mandible	21	28	24	15	19	15
Average no. of cavities per animal	11.2	19.1	16.0	4.28	8.3	7.2

Group I, hamsters, fine corn, H_2O. Control.
Group II, hamsters, fine corn, H_2O, Septochem. Full mouth.
Group III, hamsters, fine corn, H_2O, Septochem. Left side.
Group I, rats, coarse corn, H_2O. Control.
Group II, rats, coarse corn, H_2O, Septochem. Full mouth.
Group III, rats, coarse corn, H_2O, Septochem. Left side.

In each instance, the teeth to be treated were isolated by a procedure similar to that of Sognnaes (7). After the teeth were cleaned with benzine, the first "Septochem" component, presumably silver nitrate, was applied by rubbing all tooth surfaces with saturated cotton pellets for periods of 5 minutes. The second solution, presumably indigisol, was applied in a similar manner. Before the isolating appliance was removed, the excess "Septochem" was removed by washing with distilled water. The topical applications were carried out under

ether anesthesia. Animals in the control groups were also anesthetized and their teeth isolated and treated with benzine but not subjected to "Septochem" therapy. Similar isolations and treatments were given to the untreated right side of Group III hamsters and rats. Since the "Septochem" treatment causes black stains on the teeth it was readily observed that the treated surfaces were thoroughly impregnated.

At the end of 4 months the animals were sacrificed and the jaws were fixed in formalin. After the jaws had been denuded of soft tissue they were dried and examined for caries. These examinations were carried out under a binocular microscope using specially adapted probes.

FINDINGS

The findings are reported in detail in Table I and are self-explanatory. Although the groups are small it is evident that in this experiment "Septochem" did not inhibit experimental hamster or rat caries. It must be conceded that the possibility exists that more frequent applications of "Septochem" may modify this conclusion. However, it should be remembered that under similar conditions topical applications of fluoride have been shown to inhibit experimental rat (7) and hamster (8) caries.

SUMMARY

Two topical applications of "Septochem" failed to reduce the dental caries susceptibility of hamsters and white rats maintained on caries producing diets for periods of 120 days. Any effect produced was of an adverse sort.

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THE PURPOSE OF THE ODONTOBLASTS[1]

B. GOTTLIEB

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Since the odontoblasts are no longer recognized as dentin forming cells and no other function has been allotted to them (1,2), they may appear superfluous. It is not customary to find any tissue of such regular arrangement and presence which has no function. Tissues or organs which became useless during phylogenetic development are gradually reduced. In the case of the odontoblasts, neither reduction nor evidence of function in the phylogenetic past can be ascertained. On the other hand, it is known that their processes are used as invasion roads for microorganisms of caries. From that viewpoint it would be better if dentin had no fibrils around which dentinal tubuli are formed and were as solid

Fig. 1. Radiograph of 2 deciduous molars of 9 year old girl with hereditary opalescent dentin. Enamel is almost entirely lost and pulp canals are thread-like.

a material as cementum. In view of these contradictions we might consider the possibility that some useful function of the odontoblasts has been overlooked rather than blaming nature of planless construction.

It was suggested (3) that the tufts fix the enamel to the dentin. Any functioning fiber must be kept moist by tissue fluid or the functional ability will deteriorate. If one attempts to separate enamel from dentin in freshly extracted teeth with living pulps the enamel will not peel off the dentin, but if an extracted tooth is allowed to dry and ground sections are made pieces of enamel will peel off the dentin smoothly. When preparing a cavity in a tooth from which the

[1] To be presented at the General Meeting of the International Association for Dental Research, Kansas City, Missouri, March 16 and 17, 1946. Received for publication Dec. 12, 1945.

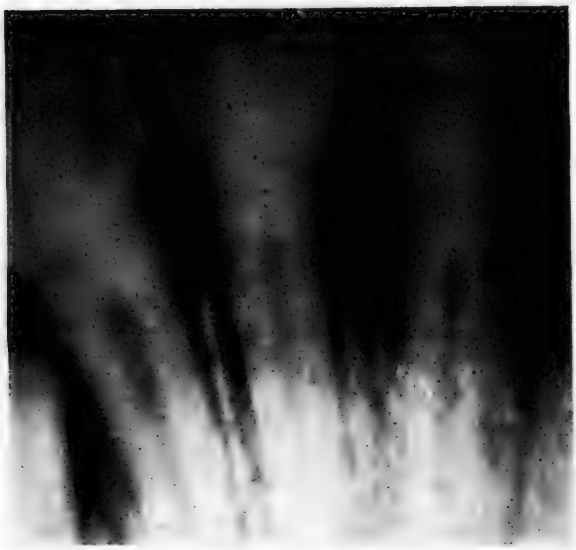

Fig. 2. Radiograph of lower incisors of child (same individual as *Fig. 1*) showing pulp chambers obliterated and pulp canal starting below level of alveolar margin. (*Figs. 1 and 2* courtesy Dr. P. F. Rosenstein, Houston, Texas.)

Fig. 3. Branching of dentinal tubules near dentino-enamel junction. A. Enamel.

pulp was removed long ago, it is sometimes surprising to find that the enamel separates from the dentin in some places. Additional information is available from cases of hereditary opalescent dentin (dentinogenesis imperfecta). Many authors (4, 5) reported, and the writer can confirm that a peeling off of the enamel from the dentin occurs quite often in such cases. *Figure 1* shows the radiograph of 2 deciduous molars of a 9 year old child with hereditary opalescent dentin. Almost every trace of enamel is lost and the pulp canals were reduced to threads. *Figure 2* shows the recently erupted lower incisors. The pulp chambers in the crown are entirely obliterated and the pulp canals start below the alveolar crest.

All these observations point to one common factor. The tufts were not moistened, dried out and failed to perform their function. In the case of the pulpless tooth and in the case of hereditary opalescent dentin, tissue fluid was not carried from the pulp to the dentino-enamel junction. Hereditary opalescent dentin is described as having only a few irregular tubules in the bulk of the dentin around the pulp (4, 5). It seems that the flow of the tissue fluid through the dentinal tubules (6) is designed to keep the tufts and the prism sheaths moist to maintain their functional ability. If that flow stops, especially in young persons, the fixation of the enamel to the dentin becomes reduced and chipping off of enamel results.

CONCLUSION

The purpose of the odontoblasts seems to be to keep open a drainage system from the pulp to the enamel. Wherever dentinal fibrils are present no dentin matrix can form and a tubular drainage system is provided. From that viewpoint it appears that the dentinal tubules branch before reaching the enamel (*fig. 3*) to permit them to act like a shower to moisten the dentino-enamel junction.

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THE FORMATION OF SECONDARY DENTIN AND RELATED PROBLEMS[1]

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FORMATION OF LOCALIZED SECONDARY DENTIN

It is generally believed that the formation of secondary dentin under fillings, caries and other forms of exposure of dentin is caused by stimuli which reach the pulp through the dentinal fibrils. It is known that the odontoblasts do not form dentin. Accordingly these stimuli must be transmitted from the odontoblasts to the surrounding connective tissue which is the source of dentin formation.

Since, at least the first formed layer of secondary dentin, in most cases, has either fewer tubules than primary dentin or no tubules at all it is difficult to explain further reaction through this new dentin. If the stimulation of the fibers is the activator for the formation of secondary dentin it can not be formed in considerable thickness once the first atubular layer is deposited. *Fig. 1*, a photomicrograph of a ground section through a tooth which was markedly abraded, shows tubules that were present and became opaque by filling in, probably with calcium salts from outside. The light area (B) is secondary dentin without tubules. There was no possibility of conducting stimuli through odontoblastic processes to the pulp in such a case, once the first layer of atubular dentin has been deposited. Nevertheless formation of secondary dentin went on to a considerable degree. In *fig. 2*, a decalcified section, the primary dentin shows regular arrangement of dentinal tubules. Beneath, is a thick layer of practically atubular dentin. If a stimulus to the pulp is necessary for formation of secondary dentin no way can be found for the continuous transmission of stimuli in formation of thick layers.

Wherever secondary dentin is deposited in a limited area due to some peripheral irritation the corresponding odontoblasts appear damaged. They are either smaller than the neighboring undamaged odontoblasts or hardly existing at all. Recently (1) this was reported from dog experiments and the same observation can be made in human beings.

The odontoblastic processes keep open drainage canals for the flow of tissue fluid to the dentino-enamel junction. The odontoblatsts must be arranged at the inside of the dentin in order to be able to continue into the dentinal fibrils. This arrangement results in them forming a separation wall between the dentin

[1] To be presented at the General Meeting of the International Association for Dental Research, Kansas City, Missouri, March 16–17, 1946. Received for publication, Dec. 12, 1945.

and the connective tissue by which the dentin was formed. The original ability of the pulpal connective tissue to form dentin seems to be greater than would

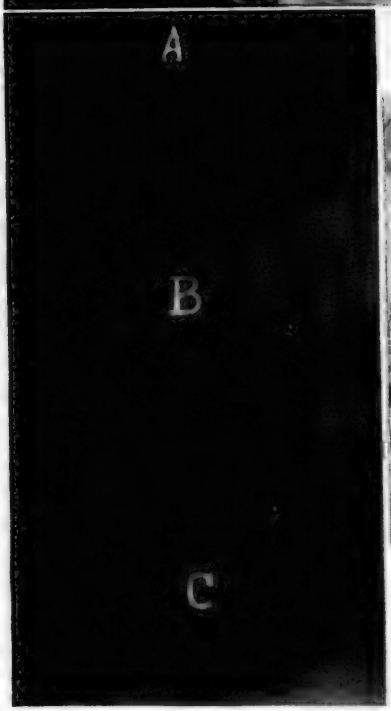

Fig. 1. Ground section showing abraded tooth with opaque tubular dentin (A), atubular secondary dentin (B) and pulp (C).

Fig. 2. Decalcified section showing primary dentin (A), atubular secondary dentin (B) and pulp (C).

Fig. 3. Decalcified section showing caries passing through tubules from primary (A) to secondary (B) dentin.

appear from causal observation. The separating layer of the odontoblasts seems to slow down the activity of the connective tissue. If however the odontoblasts

are damaged, by injury to their fibrillar extensions, their separating function is diminished. There are various degrees in which damage to the odontoblasts appears, they can become smaller as compared with neighboring undamaged odontoblasts or they may disappear. The number of fibers available for providing tubules depends on the degree of damage. Some tubules may continue without interruption from the primary into the secondary dentin. They may be used as tracts for spreading of caries between the two kinds of dentin. *Fig. 3* shows the spread of caries from the primary to the secondary dentin through continuous tubules. The same may be observed in the spreading of silver nitrate (1).

The flat cells lining the dentin in *fig. 2* appear to be undifferentiated fibroblasts. In such regions of damaged odontoblasts the connective tissue comes into better contact with the dentin and forms more matrix which shows up as localized increase of dentin formation. Thus, *secondary dentin is not formed by creating a stimulus for, but by removing a barrier against, increased deposition of new dentin matrix.*

OBLITERATION OF THE PULP CANAL BY EXCESSIVE DENTIN FORMATION

This viewpoint is also valid in the cases of filling in of the entire pulp canal by increased amount of dentin formation. We find it in several instances, the 2 most common being ageing and trauma. It must be understood that the odontoblasts are higher specialized cells as compared with the ordinary connective tissue cells. They appear to age earlier. Numerous instances can be found in teeth of adults and old people where the odontoblasts have partially or entirely disappeared. The subsequent course of development depends on the condition of the connective tissue. If the odontoblasts vanished at a time when the connective tissue was still capable of function, an obliteration of the pulp by filling in of dentin will develop. If however the connective tissue is already degenerated at that time the size of the lumen of the pulp will remain static.

A similar consideration is true under trauma. Obliteration of the pulp canal can often be observed in a traumatized tooth. If the pulp at the apical entrance was not so severely damaged as to produce necrosis of the pulp the connective tissue may recover while the higher specialized odontoblasts do not. This situation causes a general increased amount of dentin formation, leading to obliteration of the pulp canal. *Fig. 4* shows the radiograph of a traumatized lower incisor with a healed fracture at the apical third indicated by the bend in the root. The pulp canal is almost entirely obliterated as compared with the 2 neighboring teeth. The bone near the fracture area shows some pathosis.

Another possibility of obliteration of the pulp canal is that in hereditary opalescent dentin (dentinogenesis imperfecta). It was reported (3, 4) that regular arrangement of the tubules is only to be found in the peripheral strip corresponding roughly to the mantel dentin, while the main bulk of the dentin, centrally, is practically atubular. The absence of tubules means absence of dentinal fibrils, and, in turn, absence of odontoblasts. The predominant characteristic of hereditary opalescent dentin appears thus to be a systemic early de-

generation of the odontoblasts. The background of that systemic degeneration is not known. The absence of the odontoblasts induces more dentin formation leading to the obliteration of the pulp canal. The absence of the tubuli makes moistening of the enamel tufts impossible and the enamel chips off from the dentin.

Fig. 4. Radiograph of traumatized lower incisor with obliterated pulp

Fig. 5. Radiograph of traumatized upper incisor with obliterated pulp and resorbed apex.

INTERNAL AND EXTERNAL RESORPTION

In *fig. 4* the result of trauma with obliteration of the pulp canal is seen. In the tooth in *fig. 5* trauma caused obliteration of the pulp canal and, in addition, resorption of the apical end of the root. It appears strange that the same external cause produces 2 extremely different results. In orthodontics it has been shown that resorptions, mostly at the apical end, may be caused by the traumatic influence of the treatment.

Fig. 6 shows extensive internal resorption apparently caused by orthodontic treatment continued over a 6 year period in a young individual. Thus the situation becomes more complicated. Trauma of a tooth may cause obliteration of the pulp canal, resorption at the pulp walls or resorption at the root surface.

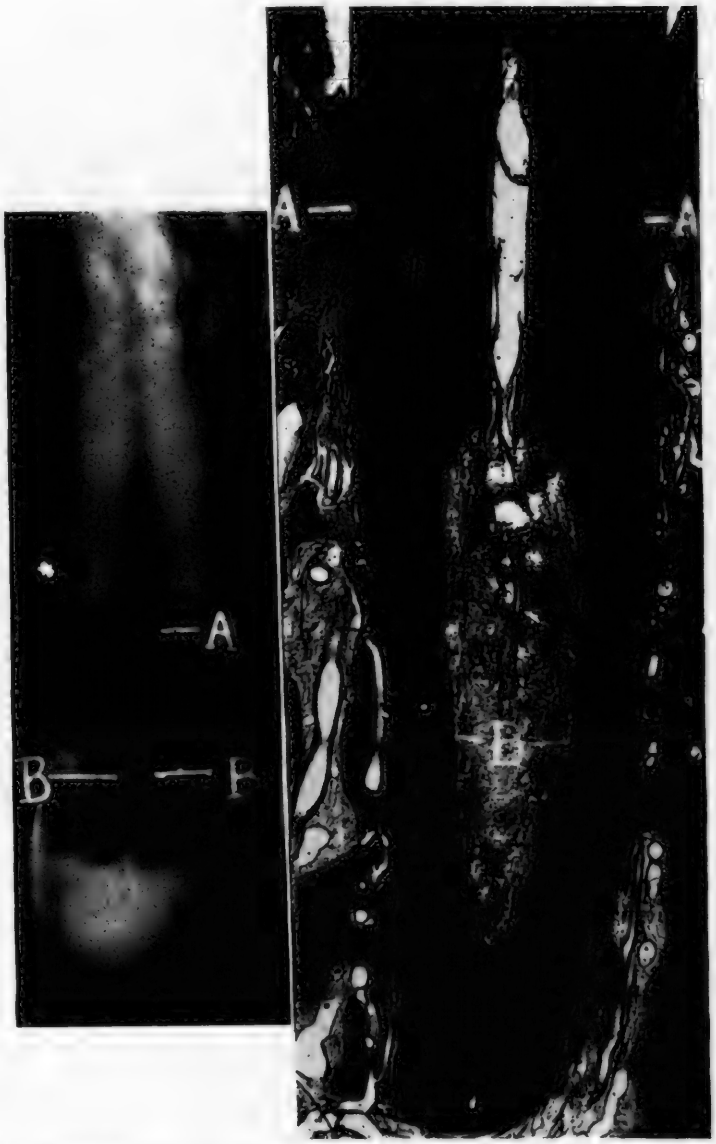

Fig. 6. Radiograph of tooth with internal resorption. A. Point of probable start of resorption from pulp. B. Circumpulpar dentin resistant to resorption.

Fig. 7. High voltage electric current passed through neighboring papilla. (A) External resorption, (B) Internal resorption.

Fig. 7 shows the result of an experiment in a dog. A high alternating electric current was applied to an interdental papilla for several seconds. The dog died

a few months later. The teeth on both sides of the papilla showed internal resorption. In addition, the tooth shows external resorptions on both sides. Both internal and external resorption were caused by the same electrical trauma. The tooth showed minute early deposition of hard tissue in some places of internal resorption. Another case of internal resorption observed in a human tooth showed considerable bone deposition in the place of the past resorption.

From numerous observations we know that external resorptions may stop at any place and repairing deposition may start. This indicates that only a limited portion of hard tissue is resorbed. When the limits of that part are reached resorption stops and deposition may start. Some time ago it was suggested (5) that some quality of the hard tissue may determine the start and extent of resorption. No detailed suggestion as to the nature of that quality could be made. It was suggested that it consists of a deterioration. A number of observations could be explained that way. The problems mentioned here belong in the same category.

In the tooth shown in *fig. 7* the electric trauma damaged some parts of the hard tissue at the surface and at the pulp walls and here resorption developed. These may have been the places through which the current passed. Resorption continues until all damaged hard tissue is removed. In the tooth in *fig. 5* the hard tissue at the apex was damaged, and in the tooth in *fig. 6* a part around the pulp was injured. In the latter case (*fig. 6*), the entrance to the resorbed dentin from the pulp seems to have been at the darkest place of the canal in the radiograph. The resorption developed towards the crown leaving a thin circumpulpal wall of dentin unresorbed. That is the most recently formed portion of dentin and it has been shown (6) that it resists more resorption than any other part.

SUMMARY

Deposition of dentin in the pulp canal seems to depend on the degree to which the separating layer of the odontoblasts is able to slow down dentin formation. If the odontoblasts are damaged in a limited area, localized increase of secondary dentin formation results. If the odontoblasts are entirely gone while the connective tissue of the pulp is in good functional condition a general accelerated formation of dentin develops, leading to obliteration of the pulp canal.

External and internal tooth resorption seems to depend on the extent to which the hard tissue has been altered. Nothing is known of the quality of that alteration.

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NUTRITIONAL REQUIREMENTS FOR AN ORAL STRAIN OF LACTOBACILLUS ACIDOPHILUS[1]

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INTRODUCTION

The relation of acid production by bacteria present in the oral cavity to the etiology of dental caries has received attention for a long period (1). More recently a number of publications have described the kinds of aciduric organisms in oral cavities (2, 3), while others have assayed the frequencies with which various species occur (4, 5, 6).

Considerable information has been accumulated regarding the nutritive factors necessary for the growth of bacteria of various species (7–12). For some organisms, such as *Lactobacillus casei*, it has been shown that the quantity of acid produced is correlated with the process and the extent of growth (13). This relation has become the basis of analytical methods for measuring the quantities of certain vitamins (14) and amino acids (15) in artificial media.

Quantitative data regarding the chemical factors necessary for the nutrition of any of the aciduric organisms present in the oral cavity are almost nonexistent. Hill and Kniesner (16, 17) have stated that pantothenic acid and dextrose are necessary for the growth of *Lactobacillus acidophilus*. These are the only reports on the nutrition of oral bacteria of which we are aware. Therefore, it seemed desirable to acquire as much information as possible regarding the growth requirements of a typical microorganism from the human mouth for any contribution which such a study might make to the important problem of dental caries.

It was thought too, that such an investigation might lead to information as to the usefulness of this microorganism in biochemical assay.

For the initial study the organism selected was *Lactobacillus acidophilus*. The strain used has been identified bacteriologically and has been grown repeatedly on synthetic media in which the constituents are largely known chemical substances.

ISOLATION AND IDENTIFICATION OF THE MICROORGANISM

The microorganism used was obtained from the mouths of clinic patients having active dental caries. Each person was given paraffin to chew and the saliva secreted in response to this stimulus was collected at once in a sterile beaker. A loop full of the well mixed specimen was plated directly on a 1% tryptic digest agar of pH 5.0. A 1% dextrose broth (pH 5.0) was inoculated with a similar

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portion of the same sample. Both were incubated at 37.5°C. for 24 hours. The dextrose broth was then streaked on 1% tryptic digest agar. Following incubation, small fluorescent colonies of pleomorphic, Gram positive bacilli were fished to sugar fermentation tubes and incubated at 37.5°C. for 24 hours. The results of these tests are recorded in Table I.

The morphology of this strain and its fermentation reactions are similar to the findings of Morishita (18), who studied strains of aciduric organisms isolated from carious teeth and saliva, as well as to those of Enright, Friesell and Trescher (19) who studied the characteristics of lactobacilli isolated from 68 specimens of human saliva.

Hereafter the strain isolated and used in these studies shall be designated as *Lactobacillus acidophilus* 333. Stock cultures of the organism were maintained in a broth medium.[2]

TABLE I

Action of Lactobacillus acidophilus Strain 333 on various polysaccharides

SUGAR	ACID FORMED	SUGAR	ACID FORMED
Dextrose	+	Sucrose	−
Levulose	+	Raffinose	−
Galactose	+	Mannitol	+
Arabinose	−	Sorbitol	+
Xylose	−	Salicin	+
Maltose	+	Dextrin	+
Lactose	+	Insulin	−

EXPERIMENTS WITH ENRICHED MEDIA

In some experiments the quantities of individual constituents present was varied in order to gain information regarding their potential rôle in the growth of the microorganism. The preparation of the media for these tests may be illustrated by a description of the experiment in which the quantity of casein hydrolysate was the variable. The double strength media was made up in accord with the specifications given in Table II except that casein hydrolysate was omitted. Then, casein hydrolysate was added to appropriate tubes in 3 different amounts. The tubes were adjusted to pH 6.8, made up to volume with distilled water, mixed and autoclaved. Later they were inoculated with equal quantities of the test microorganism. Similar procedures were used in other experiments for varying the quantities of other constituents of the media.

Preparation of Media. The basal medium employed in these experiments was that described by Landy and Dickens (14). As used here 50 ml. of double strength media contained the substances and quantities listed in Table II.

[2] (Consisting of 2.5 ml. of casein hydrolysate (S.M.A.Co.), 0.21 ml. of Liver Extract (Lilly), 0.25 ml. of salt mixture A, 0.25 ml. of salt mixture B (Landy and Dickens (14)) 300 mg. of sodium acetate, dextrose to make a 1% solution and solution and distilled water to 50 ml.

The individual substances were maintained in stock solutions in a refrigerator. For use in experiments necessary quantities of each were transferred to a 50 ml. volumetric flask and the combination was made up to volume with distilled water. Five ml. of the double strength media thus prepared was transferred to Pyrex

TABLE II

*Chemical Composition of Basic Medium**

Casein hydrolysate[a] (Vitamin free)	2.5 ml.
Salt[b] mixture A†	0.5 ml.
Salt[b] mixture B‡	0.5 ml.
Glucose[c]	1 gm.
Sodium acetate[b]	600 mg.
Asparagine[d]	25 mg.
Tryptophane[e]	10 mg.
Cystine[e]	10 mg.
Guanine hydrochloride[e]	500 γ
Adenine sulfate[e]	500 γ
Xanthine[e]	500 γ
Uracil[e]	500 γ
Pyridoxine hydrochloride[b]	40 γ
Riboflavin[b]	20 γ
Calcium pantothenate[b]	20 γ
Nicotinic acid[b]	20 γ
Thiamin hydrochloride[b]	10 γ
Folic acid	1 γ
Biotin[a]	0.5 γ
Distilled water to 50.0 ml.	

Sources of Materials Used in the Media

[a] S. M. A. Co.
[b] Merck and Co.
[c] Baker Chemical Co.
[d] Pfanstiehl Chemical Co.
[e] Eastman Kodak Co.

* Double strength.

† Salt Mixture A

K_2HPO_4	5 gm.
KH_2PO_4	5 gm.
Water	50 ml.

‡ Salt Mixture B

$MgSO_4 \cdot 7\ H_2O$	10	gm.
NaCl	0.5	gm.
$FeSO_4 \cdot 7\ H_2O$	0.5	gm.
$MnSo_4 \cdot 2\ H_2O$	0.337	gm.
Water	250	ml.

culture tubes and the test material was added in a solution to the appropriate concentration. The pH was adjusted to 6.8 by the addition of 0.1 N. sodium hydroxide and then finally sufficient distilled water to dilute to 10 ml. of single strength media was added. The contents were well mixed, the tubes plugged and autoclaved for 10 minutes at 20 pounds pressure.

Technique of tests. The inoculum was prepared from a fresh culture of Lactobacillus strain 333 grown for 18 hours in a fresh portion of the broth medium described above. At the end of this period 7 ml. of the well suspended culture was centrifuged and the supernatant liquid removed aseptically. The sediment was resuspended in 7 ml. of sterile normal saline. One tenth ml. of the thoroughly mixed suspension was added to the appropriate media tubes by means of a sterile 1 ml. microburette with the precautions necessary to provide uniformity in the quantity of organisms delivered.

The tubes containing the test materials were incubated for 72 hours at 37.5C. Then the contents of each tube in turn were rinsed into 50 ml. beakers with the aid of a few ml. of distilled water. The acid formed by the microorganism was

TABLE III

Effect of Variations in Composition of the Constituents of the Medium on Acid Production

CONSTITUENT	QUANTITY ADDED	ACID PRODUCED	QUANTITY ADDED	ACID PRODUCED	QUANTITY ADDED	ACID PRODUCED
		ml.		*ml.*		*ml.*
Casein hydrolysate	0.5 ml.	4.86	1.0 ml.	4.91	1.5 ml.	4.91
Salt mixture A	0.05 ml.	4.91	0.5 ml.	4.90		
Salt mixture B	0.05 ml.	4.78	0.5 ml.	4.84		
Asparagine	1.0 mg.	4.91	2.0 mg.	4.96		
Cystine	1.0 mg.	4.84	2.0 mg.	4.79		
Tryptophane	1.0 mg.	5.02	2.0 mg.	4.97	4.0 mg.	4.98
Guanine hydrochloride	50 mg.	4.77	100 mg.	4.68		
Adenine sulfate	50 mg.	4.83	100 mg.	4.76		
Xanthine	50 mg.	4.89	100 mg.	4.80		
Uracil	50 mg.	4.69	100 mg.	4.81		
Thiamin hydrochloride	1 γ	4.87	2 γ	4.89	3 γ	4.77
Riboflavin	2 γ	4.95	4 γ	5.01	6 γ	5.00
Calcium pentothenate	2 γ	4.84	4 γ	4.91	6 γ	4.78
Nicotinic acid	2 γ	4.77	4 γ	4.84	6 γ	4.76
Folic acid	1 γ	4.86	3 γ	4.77		
Biotin	0.05 γ	4.88	0.5 γ	5.01	1.0 γ	4.74

The quantities specified are those present in 10 ml. of the media.

titrated to a pH of 6.8 with 0.1 N. sodium hydroxide using a Coleman pH Electrometer to locate the end point.

Results. In a series of such experiments, microorganisms which were isolated from the mouths of 23 patients and having the characteristics of strain 333, grew readily and abundantly in the synthetic media described by Landy and Dickens (14). In these experiments the acid yielded was sufficient to neutralize 4.2 to 5.0 ml. of 0.1 N. base. Since this observation has been repeated many times, it seems established that this strain of oral bacteria can be grown in a highly purified medium.

Additional experiments were made to determine whether the medium of Landy and Dickens (14) was adequate for providing maximum growth or whether enrichment of some of the constituents would increase growth, as

assayed by production of acid. The preparation of media in which the concentration of each constituent in turn could be varied has been described above. Using this preparation a series of tubes in which each constituent was increased in concentration, were prepared. Each was inoculated with the same volume of strain 333, and incubated at 37.5°C. for 72 hours. Then the total quantity of acid produced in each was determined. The results obtained are presented in Table III. The smallest (column 3) quantity listed for a particular substance being tested is that present in the media made according to the specification of

TABLE IV

Determination of Constituents in Basal Medium Essential to Maximum Growth

NUTRIENT SUBSTANCES*	AMOUNT (ML.) OF ACID PRODUCED IN BASIC MEDIUM	
	with	without
Essential		
Thiamin hydrochloride	4.83	0.00
Nicotinic acid	4.79	0.00
Calcium pantothenate	4.92	0.00
Tryptophane	4.88	0.00
Casein hydrolysate	4.91	0.00
Salt mixture A	4.90	0.00
Non-essential		
Riboflavin	4.89	4.89
Biotin	4.82	4.92
Pyridoxine hydrochloride	4.88	4.88
Folic acid	4.73	4.76
Asparagine	4.69	4.68
Cystine	4.50	4.52
Xanthine	4.70	4.77
Adenine sulfate	4.81	4.87
Guanine hydrochloride	4.80	4.72
Uracil	4.78	4.78
Salt mixture B	4.86	4.85
Essential substances only	4.83	
Complete media	4.88	

* d glucose present in each experiment.

Landy and Dickens (14). The small magnitude of the differences shown in this column is an indication of the extent to which constant experimental conditions were reproduced in the successive trials. The variations in the quantities of the individual constituents being tested for their enhancing effect ranged from a 10% increase of some substances to a 100% increase of others. In no instance was the increase in the individual constituent of the media accompanied by any significant increase in the quantity of acid produced. Therefore, it appears that the original media of Landy and Dickens (14) contains the constituents necessary for the satisfactory growth of this strain of *Lactobacillus acidophilus*. Also the

quantities for each constituent specified by these authors are sufficient to yield maximum growth of this organism.

MINIMUM GROWTH REQUIREMENTS

The essentiality of the various constituents of this basic medium for maximum growth was next determined in order that a simplified medium consisting only of the essential growth substances could be prepared. The essentiality was determined by the preparation of different lots of basic medium from which a basic substance was omitted. The preparation of the medium may be illustrated by a description of the experiment in which casein hydrolysate was omitted.

The tubes were adjusted to pH 6.8, made up to volume with distilled water, mixed and autoclaved. Later they were inoculated with equal volumes of the test microorganism. Similar procedures were used in other experiments, each omitting one of the constituents of the basic medium. Lastly the microorganisms were grown in a medium consisting only of the substances found to be essential. The technique of the tests were identical with those already described.

Results. It was demonstrated by these elimination experiments (Table IV), that maximal acid was produced by the test microorganism, even when the following known substances were omitted from the complete medium one at a time; asparagine, cystine, guanine hydrochloride, adenine sulfate, xanthine, uracil, riboflavin, pyridoxine hydrochloride, folic acid, biotin, salt mixture B. A simplified medium was then employed in which all of the substances found to be non-essential were omitted. When this oral strain of lactobacillus was grown in such simplified medium containing only the known essential substances, maximal acid was produced. Such a simplified medium although supplying sufficient nutritive substances for maximum growth of the organisms may, however, be deficient in enhancing substances which, if present, might support an earlier maximal growth. However, the present purpose was only to determine those nutritive substances which are essential for maximum growth.

SUMMARY

Specimens of *Lactobacillus acidophilus* isolated from the mouths of 23 clinic patients having active dental caries have been shown to have uniform morphological and biochemical characteristics. This organism has been grown successfully on the medium of Landy and Dickens which consists almost entirely of purified chemical substances. The quantity of acid produced in standardized experiments was closely reproducible. Enrichment of the media by addition of extra quantities of each individual constituent was not accompanied by any significant increase in the production of acid. Omission of certain of these constituents caused almost complete cessation of acid production while others have been found to be non-essential.

A medium consisting only of glucose, tryptophane, sodium acetate, potassium phosphates, thiamin hydrochloride, calcium pantothenate, nicotinic acid and

vitamin free casein hydrolysate is capable of providing for maximum acid production with this oral strain of *Lactobacillus acidophilus*.

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ORDER AND AGE OF ERUPTION FOR THE DECIDUOUS DENTITION[1]

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Presently available research literature includes no less than a score of North American studies pertaining to the time and sequence of eruption of the primary or "milk" teeth. For only about one-sixth of these studies is the medium of original report a dental periodical, the remaining five-sixths are in anthropological, biological, child development, medical (mainly pediatric) and psychological publications. It is a major objective of this paper to bring together these widely scattered reports and make their contents more readily accessible to members of the dental profession.

A second major objective of the paper is that of presenting an epitome of research knowledge concerning the age and order of eruption of deciduous teeth in North American children. This, of course, presupposes a systematic and comprehensive integration of the materials from different studies.

The two objectives determine the two parts of the paper: The first—colligation—is carried out under the caption "Presentation of Research Materials" and the second—synthesis—under the caption "Generalizations." There are incorporated in both parts the findings from two previously unpublished investigations.

PRESENTATION OF RESEARCH MATERIALS

As a point of departure, reference may be made to a "Course of lectures on dentition and its derangements" published by Jacobi (9) in 1860. The materials in these lectures pertaining to eruption of the deciduous teeth were drawn entirely from European studies.

Minot (13), in 1873, wrote a paper "On the primary dentition of children" which included statements relating to the order and age of eruption based upon his "own experience." On eruption of the first tooth, Minot stated: "Judging from my own observation, I should put six and a half months as the average date of the appearance of the first tooth" (13, p. 9). The usual sequence of eruption for the full complement of deciduous teeth was given as follows: The "two lower central incisors" at 6½ to 7 months; "The four upper incisors, of which the two central ones are the first to appear," at 9 to 10½ months; the "upper anterior molars," lower lateral incisors, and "lower anterior molars"—in this order—at 12½ to 14 months; "the four canines" at 18 to 21 months; and "the four posterior molars" at 26 to 30 months.

[1] Received for publication January 14, 1946.

At the 1876 annual meeting of the American Dental Association, McQuillen "made a verbal report on the order of eruption of the deciduous teeth" (11, p. 483). After commenting that instances were on record of infants "having been born with teeth," he stated: "The central incisors usually erupt from the sixth to the eighth month, and the laterals from the seventh to the ninth month; the first molars from the fourteenth to the sixteenth month; the second molars between the twenty-fourth and thirtieth months" (11, p. 484). No mention was made of the typical age of eruption of the deciduous canines, nor was there any reference to the source of the information on other teeth. McQuillen's report was discussed by a "Dr. Shepard," who presented "a case of variation in the time of eruption of the deciduous teeth in the same family; one boy had erupted a tooth at four months of age, and at eight months had eight teeth, while another had no teeth till eight months old, and at one year had but five teeth" (11, p. 527).

Shinn (20), in 1893, published "notes" on the eruption of the "milk" teeth in a single female infant. The subject was a physically normal infant born and reared "on a fruit ranch near Niles, in the neighborhood of San Francisco Bay" (20, p. 5). She was of 'Old American' stock, "all four grandparents being descended from early colonists" (20, p.5). Shinn observed: ". . . two teeth were cut just after the close of the fifth month. In the ninth and tenth months six more teeth were cut. . . the first molar came through early in the fifteenth month" and "teething was distributed quite equally" throughout the ensuing months "to the end of the nineteenth" (20, p. 8). "In the latter part of the twenty-seventh month, two more molars were cut" (20, p. 89). It may be inferred that at 1 year of age the 8 erupted teeth were incisors, by 2 years of age the first molars and canines had erupted, and the teeth erupting in the third year were second molars.

In 1895, a paragraph describing original data for eruption of the first tooth in New York City infants was printed in *The Dental Review* and reproduced in *Ash's Quarterly Circular* (1). To quote: "With the aid of Dr. Purdy, Dr. J. Lewis Smith has kept records of the average time of appearance of the teeth in children at the out-door department of Bellevue Hospital, excluding cases of rickets, which were most common in Italians and next in Negroes. In 200 infants without signs of rickets, the first tooth had appeared as follows: In 3 infants at two months, in 20 at five months, in 24 at six months, in 37 at seven months, in 28 at eight months, in 20 at nine months, in 14 at ten months, in 15 at eleven months, in 8 at twelve months, and in 1 at thirteen months of age" (1, p. 203). Statistical reduction of these data yields the following tabulation:

Age at Eruption of First Tooth (mos.) in New York City Infants

MINIMUM	PERCENTILES			MAXIMUM
	25	50	75	
2	5.8	7.1	8.8	13

The mean age at eruption was 7.3 months, the median age (as above) 7.1 months.

Hrdlicka (8), in 1908, reported observations for deciduous eruption obtained

on American Indian children of the Pima and Apache tribes of Arizona. For "full-bloods" of "known age," there were 120 subjects distributed from birth to 6 years—between the ages of 2 months and 2½ years the numbers were 35 Pima (14 males, 21 females) and 35 San Carlos Apache (17 males, 18 females). The data were collected from single examinations made between 1898 and 1905. Hrdlicka noted that it was customary practice among these tribes for infants to be at least partly breast fed until after 2 years of age. Other statements pertaining to diet were: "For the first five or six months the Apache baby gets only mother's milk; later on it receives a little food of any kind eaten by the mother" (8, p. 76); the Pima infant, "in addition to being nursed," is sometimes fed "on fruit and other things from the time it is about four or five months old" (8, p. 78).

"Among the San Carlos Apache the youngest child with one or more teeth was a girl in whom both lower middle incisors appeared at 2½ months, and among the Pima a girl who had both lower median incisors fairly developed at 4 months and 13 days. Among the Apache the oldest infant without teeth was a girl of 7 months, and among the Pima a boy of 7 months and 20 days.

"The upper median incisors follow the lower. . . .among the Apache the youngest child in which both these teeth were erupted was 7 months and 20 days, among the Pima 7 months and 17 days, but in both children the teeth had been out for some days . . . the oldest subjects in whom the eruption of the upper median incisors had not taken place were a 12½ months Apache girl and a Pima girl of the same age.

". . . the upper lateral incisors. . . had just broken through in an Apache girl of 7 months and 20 days and in a Pima boy of 7 months and 17 days. . . the oldest children in whom both were still lacking were an Apache gril of 14 months and a Pima girl of 1 year and 16 days old.

"The lower lateral incisors. . . were seen earliest in an Apache girl of 8½ months and in a Pima girl of 9 months. The oldest Apache girl in whom both were lacking was 12½ months; the oldest Pima child 18 months and 2 days old. In three instances one or both of these teeth were out before the upper lateral incisors.

"Among the Apache children in two cases all the anterior premolars were out before the appearance of any of the canines, and there are indications of similar precedence among the Pima. . . .As to the time of appearance, in the Apache the anterior premolars were not present in any child up to 14 months, but were found in all of 16½ months and older, while in the Pima the teeth were not erupted in any subject up to 15½ months, and were present in all but one of the children of 17½ months and older.

"The canines were seen first in an Apache girl of 19 months 10 days, all being present in every older child, and in a Pima girl of 17½ months, being present in all but two older subjects (of 18 months and 18 months and 2 days respectively).

"The posterior premolars. . . were all present in an Apache child of 19 months 10 days, and in another of 20 months; these had not all erupted in three Indian children of from 22 to 23 months of age and were wholly lacking in one of 25 months, but were present in every case from 26 months upward. Among the Pima none of these teeth were found in children 2 years or younger; the two lower were present in a boy of 25 months, and were just appearing in another of 26 months 9 days, but were present in every subject above this age" (8, pp. 96–97).

It was recognized by Hrdlicka that for each tribe and sex the number of children studied was "very limited."

In 1913, Herrman (7) published eruption data for the "first teeth" collected on infants born "June 1, 1909, to June 1, 1910" at Lebanon Hospital, New York. The subjects were approximately 50% of Russian-born parentage, 30% of north-

west European ancestry, and 20% of southeast European descent. Their mothers took them to the Hospital Dispensary at frequent intervals over the first 2 years of postnatal life; here, the infants were examined and the mothers given advice on dietary and health care. The sample seemingly included "careless" as well as conscientious mothers, and fairly sick as well as healthy infants. Over four-fifths of the subjects "received breast milk exclusively or partly for at least five months" (7, p. 110).

Eruption Age for "First Teeth" (mos.) in White Infants Studied 1909–11

N	MEAN	S.D.	MINIMUM	PERCENTILES		MAXIMUM
				25	75	
121	8.2	1.8	4.4	7.0	9.2	12.5

Discussion of findings was restricted to a single notation: "Seventy per cent had their first teeth at or before eight and a half months" (7, p. 109).

Two series of data representing eruption of the deciduous teeth in children living in an institution—"The Home for Hebrew Infants, New York City"—were analyzed in 1927 by Boas (2). For one series, the procedure was as follows: Children were "observed at intervals of three months by Dr. Louis N. Sullivan and Mr. Maynard. . . .The teeth were recorded as absent, erupting or fully erupted. . . .The age at the time of eruption was assumed as the middle date between the last date of absence of a tooth and the first date of its full presence. In cases of erupting tooth, twenty days were added to the date of the observation when eruption was recorded" (2, p. 245). The analysis for these materials is presented in the upper portion of Table I.

The second series of data was characterized as "observations of presence or absence" for deciduous teeth "at given ages." In this series, it was possible to include "a much larger number" of subjects (*e.g.*, "children who were just about to be discharged from the Infant Home"): Specifically, the average number examined at each month of age was 20 for males and 13 for females. There was separate treatment of the data on each sex. The lower portion of Table I reproduces findings from reduction of these materials.

The central tendency values (medians and means) from Table I were discussed with reference to order of eruption of teeth, the means with reference to sex differences in age of eruption, and the standard deviations with reference to their magnitude for different teeth. To quote: "The variability is least for the inner incisor, and reaches its maximum for the second deciduous molar" (2, p. 253); ". . . it would seem that there is no characteristic sex difference in the time of eruption for these children" (2, p. 253); ". . . for the inner incisor there is the well known characteristic difference in favor of the lower jaw. . . . There is also a slight difference in favor of the lower jaw in the case of the second deciduous molar. . . . It seems likely that the outer incisor of the upper jaw precedes slightly in eruption the corresponding tooth of the lower jaw" (2, p. 253). Boas further stated: ". . . it seems that the Home children are very much retarded in the eruption of deciduous teeth . . . possibly this is due to the poor condition in which most of the children are when taken to the Home" (2, p. 252).

Sawtell (19), in 1929, reported findings for eruption of the deciduous dentition

on two socio-ethnic groups. One group consisted of "twenty-eight Sicilian infants from the lower East Side of New York"—"fourteen boys, average age 9.1 months, fourteen girls, 8.1 months" (19, pp. 62, 73). Eruption data on these subjects were made available by Mr. John C. Gebhardt. Sawtell found that the mean number of "incisors present" approximated 2.6 for the males and 1.4 for the females.[2] Comprising the second group were 16 males and 14 females between the ages of 30 and 42 months in attendance 1925–27 at the nursery school

TABLE I

Eruption of Deciduous Teeth (mos.) in "Hebrew Infants"

Age of eruption for both sexes combined

	N	MINIMUM	PERCENTILES			MAXIMUM
			25	50	75	
Lower first incisor*.....	45	4.5	9.3	11.1	13.4	19.5
Upper first incisor.....	53	4.5	10.8	13.1	14.8	23.5
Upper second incisor....	68	8.5	13.3	16.3	18.7	28.5
Lower second incisor....	54	8.5	14.1	16.8	18.4	30.5
Lower first molar.....	70	13.5	18.0	19.9	21.1	29.5
Upper first molar.....	80	13.5	18.0	20.2	22.0	28.5
Lower canine.....	89	13.5	19.6	21.8	24.8	34.5
Upper canine.....	97	13.5	19.2	21.8	24.9	33.5
Lower second molar....	80	14.5	24.9	27.4	30.1	40.5
Upper second molar....	82	14.5	25.3	28.6	31.1	44.5

Age of eruption for each sex separately

	LOWER TEETH				UPPER TEETH			
	Males		Females		Males		Females	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
First incisor.....	11.0	2.8	11.0	3.0	12.8	2.8	13.5	3.1
Second incisor.....	16.8	4.0	16.4	4.3	15.4	3.4	15.0	3.5
First molar.....	19.5	3.1	19.6	4.4	19.2	2.9	19.4	4.2
Canine.....	21.7	4.5	21.6	4.9	21.1	4.5	21.8	5.1
Second molar.....	28.4	4.8	28.1	4.9	28.7	5.1	28.5	4.8

* It was not specified whether the data symbolized (a) age of eruption of each tooth on one side of the mouth, (b) average age of eruption for each pair of corresponding teeth on the right and left sides, or (c) age of eruption of the first of each pair of corresponding teeth without reference to the side of the mouth on which this appeared.

of the Bureau of Educational Experiments, New York City. These subjects were predominantly of northwest European ancestry and from homes of "moderate prosperity" in the "Chelsea or Greenwich Village sections of New York" (19, p. 62). Four of the 30 (13%) showed incomplete eruption of the deciduous dentition: At 34 months of age, a boy lacked "the upper second molars" and a girl "the upper right second molar"; at 37 months, a girl lacked both upper second

[2] The reader is cautioned not to overlook the fact that the Sicilian females averaged one month younger than the Sicilian males.

molars, and a boy, "known to have an early rachitic history, . . . had only sixteen teeth" (19, p. 83).

Using the results of annual dental examinations made over a 4-year period, Pyle and Drain analyzed data for eruption of the deciduous teeth on 165 males and 175 females "in attendance at the preschool laboratories of the Iowa Child Welfare Research Station" (15, p. 152). The subjects were largely of northwest European lineage and came from "homes where information and resources for proper health and dental instruction were easily available" (15, p. 147). Group findings are reproduced in Table II.

TABLE II

Mean Number of Deciduous Teeth Erupted at Successive Ages

Data accumulated 1927–31 on Iowa City private school children

AGE GROUP		MALES		FEMALES	
Midpoint	Range	N	Mean	N	Mean
mos.	*mos.*				
24	18 thru 29	22	17.4	19	16.2
36	30 thru 41	34	19.6	49	19.6
48	42 thru 53	49	20.0	50	20.0
60	54 thru 65	60	20.0	57	20.0

"The youngest boy with a full complement was 27 months . . . the youngest girl 29 months. . . . The oldest boy who had less than twenty teeth was 35 months old and the oldest girl 37 months" (15, p. 148).

Findings for age of eruption of the first tooth, and for status of the deciduous dentition at the beginning of the third year, were published in 1931 by Wallis (22). The data on the first tooth were secured through the City and Country School, New York City: "From the registrar's information blank is the mother's

TABLE III

Eruption of Deciduous Teeth in New York "Private School" Children

Age (mos.) at which "mothers said positively" first tooth appeared

SEX	N	MEAN	S.D.	MINIMUM	MAXIMUM
Males	147	7.6	2.3	3	15
Females	130	7.6	2.4	3	16

Status of deciduous dentition in age period 22 to 27 months

SEX	N	PERCENTAGE WITH:		MEAN NUMBER OF TEETH	S.D.
		16 teeth	20 teeth		
Males	16	56	25	17.1	1.8
Females	15	40	33	16.7	3.0

statement, verified whenever possible by the baby book, of the date on which the first tooth erupted" (22, p. 129). At ages "twenty-two to twenty-seven months," 31 children were examined over the period 1926–29 at the nursery school of the Bureau of Educational Experiments, New York City. Socio-economically, these subjects were of "the favored class"; ethnically, they typified third-generation United States children of north European stocks. Table III displays the results from both analyses. Wallis recognized that the sources of unreliability were greater for the first tooth data than for the data from examination of children in the nursery school.

Eruption ages for all 20 deciduous teeth were reported by Dafoe and Dafoe (3) on each of the Dionne quintuplets. The subjects were described as female monozygotic siblings, prematurely born by approximately 2 months, of French-Canadian descent, and reared under well controlled provisions for health protection and supervision. Their dietary included breast milk until 4.5 months, tomato and orange juice from 5 weeks, cod liver oil from 8 weeks, ferrous chloride and prune juice from 4 months, cereal and evaporated milk from 5 months, egg yolk, cooked vegetables and fruit pulp from 8 months, and calves liver from 1 year. Each had a "moderately severe infection" for 1 week in the fourth month—cough, nasal discharge, fever, diarrhea, cramp and distention; mild to moderate respiratory infections occurred in the eleventh and fourteenth months; shortly before eighteen months, and again in the twenty-first and twenty-second months, each had coryza in conjunction with vomiting and loose stools. All received "three diphtheria toxoid injections at three-week intervals when $1\frac{1}{2}$ years of age" (3, p. 422). See Table IV for an itemization of the ages of eruption in each individual. Dafoe and Dafoe drew the following comparative conclusion: "The eruption times of the deciduous dentition in the quintuplets have been definitely slower than normal" (3, p. 419).

Poole, Hamil, Cooley and Macy, in 1937, studied the association between age of eruption of the deciduous teeth and "early addition of the antineuritic factor, vitamin B_1, . . . to an ordinary milk formula" (14, p. 730). The subjects were 117 full-term, non-twin, well infants "living in their own homes and cared for by their own parents" (14, p. 731). Roughly two-thirds were White and one-third Negro. They were drawn from "the indigent and near-indigent class of the cosmopolitan population of Detroit" and were studied 1934–35 through the Research Laboratory of the Children's Fund of Michigan and the Children's Hospital of Michigan. When initially examined they "ranged from 1 to 10 weeks of age, with an average of 5.5 weeks" (14, p. 748): From the time of this examination all were artificially fed. Their mothers were instructed at a clinic and by visiting nurses regarding feeding management and health care. The basic milk formula given all infants consisted of "evaporated milk, water and corn syrup acidified by the addition of commercially dehydrated lemon juice powder" (14, p. 749); cereal was added to the dietary at 4 to 5 months, strained carrots and peas at 5 to 6 months, prune pulp and apple sauce at 6 to 7 months, and eggs at 11 months; cod liver oil was given to yield 250–600 U.S.P. units of vitamin D daily. "At the first visit to the clinic, the babies were assigned . . . alternately to 2

groups"; one group received "formula containing evaporated milk to which at the time of manufacture had been added vitamin B (B_1) in the form of a water-soluble extract of rice polishings," while the other group was given a formula which "contained no vitamin B extract"; "the average estimated vitamin B_1

TABLE IV

Eruption Age of Deciduous Teeth (mos.) in 5 Prematurely Born, Monozygotic Females Studied 1934–37

	RIGHT SIDE					LEFT SIDE				
	A	C	E	M	Y*	A	C	E	M	Y
Lower teeth										
First incisor	11	12	15	15	15	14	11	15	13	14
Second incisor	16	15	16	16	16	16	14	17	15	17
First molar	20	19	18	18	19	18	19	20	18	30
Canine	23	23	24	23	24	23	23	25	23	22
Second molar	38	36	37	35	39	37	37	34	36	?†
Upper teeth										
First incisor	12	13	14	13	12	13	13	14	14	12
Second incisor	15	14	16	16	16	16	15	18	15	17
First molar	19	19	19	19	20	20	21	21	20	20
Canine	26	25	27	26	26	25	26	27	24	24
Second molar	37	35	38	36	38	36	36	36	37	38

* The initials of the children's names in alphabetical order: Annette, Cecile, Emelie, Marie, Yvonne.

† "Eruption not completed" at "40 months" (3, p. 420).

TABLE V

Mean Number of Deciduous Teeth Erupted at Selected Ages

Data accumulated 1934–35 on White and Negro infants of the lower classes fed special diets

AGE GROUP		CONTROL DIET		ADDITIONAL B_1 DIET	
Midpoint	Range	N	Mean	N	Mean
wks.	*wks.*				
18	16 thru 19	46	0.1	65	0.0
26	24 thru 27	44	0.6	61	0.5
34	32 thru 35	42	1.7	57	2.0
42	40 thru 43	36	4.4	41	3.9
50	48 thru 51	31	6.3	42	5.3

content of the supplemented formula was approximately 320 Sherman-Chase Units" and of "the non-supplemented formula was 227 Sherman-Chase units" (14, pp. 732, 733, 739). There were 66 experimental and 51 control subjects: None was under study less than 2 months; over two-thirds were followed to prac-

tically the end of the first postnatal year. The mean age at eruption of the first tooth was 7.3 months for 59 infants of the experimental series and 7.5 months for 43 infants of the control series. Findings for mean number of teeth erupted at five different ages during the first year are given in Table V. In general, the means are similar for the 2 series in the second quarter of the year, higher for the experimental series in the third quarter, and higher for the control series in the fourth quarter. The results as a whole give no dependable support to the hypothesis of a relationship between age of tooth eruption and early addition of an extract of rice polishings to the milk formula.

In 1938, Gesell and Thompson (5) published an analysis of records for number of teeth present in the mouth at different ages during the first postnatal year. The records were gathered between 1927 and 1931 at the Yale Clinic of Child Development, New Haven, Connecticut. They were obtained on 107 infants—

TABLE VI

Number of Deciduous Teeth Erupted at Selected Ages

Data on New Haven "middle class" White infants examined 1927–31

AGE	MALES				FEMALES			
	N	Minimum	Mean	Maximum	N	Minimum	Mean	Maximum
wks.								
16	25	0	0.0	0	23	0	0.0	0
20	17	0	0.1	2	19	0	0.1	2
24	16	0	0.3	2	21	0	0.3	3
28	15	0	1.0	4	18	0	0.8	6
32	15	0	2.0	5	17	0	1.5	6
36	16	0	3.4	8	18	0	2.1	7
40	20	0	4.6	8	17	0	3.3	7
44	17	0	5.5	8	16	0	4.3	8
48	18	0	6.3	10	17	0	5.5	8
52	19	4	6.4	12	24	1	5.9	10

49 males and 58 females—described as "physically normal," full-term, healthy ("Several cases of decided malnutrition were excluded"), and from "single births" (5, pp. 28–31). The parents were American-born, of "northern European extraction," and "of the middle socio-economic status with respect to occupation, schooling, avocational interests, and home equipment" (5, p. 9). Examinations were scheduled at 4-week age intervals; "some of the subjects were regularly and repeatedly examined, while others were seen only once"; "a variation of but two days from the exact age for each examination was the rule" (5, pp. 5, 9). Table VI reproduces the findings. It will be seen that the mean number of erupted deciduous teeth is greater for males than for females at all ages after 6 months.

Tooth eruption during the first year of postnatal life was studied by Hamil, Reynolds, Poole and Macy (6) utilizing a sample of 427 Detroit infants (approximately two-thirds White and one-third Negro) fed a prescribed diet from early infancy. Collection of the data was carried out 1933–36 through the resources

of the Children's Fund of Michigan and the Children's Hospital of Michigan, Detroit. The subjects were infants of "indigent or near-indigent parents" living under home conditions: All were enrolled in the study before 3 months of age and 80% were followed over " a period of nine months or more" (6, p. 566). Their dietary regimen was "as rigidly controlled as possible"—visiting nurses "kept the mothers supplied with the essential materials for the formulas and supervised the care of the infants" (6, p. 567). The basal formula was "a mixture of evaporated milk, water and corn syrup . . . acidified by the addition of commercial dehydrated lemon juice of known ascorbic acid content" (6, pp. 567–8). After

TABLE VII

Eruption of Deciduous Teeth in Infants of Lower Classes Fed a Prescribed Diet

(Data amassed 1933–36 on Detroit White and Negro infants)

Mean number of teeth at successive ages in total sample and a subgroup

AGE GROUP		TOTAL SAMPLE		MILD SCURVY SUBGROUP	
Midpoint	Range	N	Mean	N	Mean
wks.	*wks.*				
10	8 thru 11	402	0.01		
14	12 thru 15	393	0.02		
18	16 thru 19	393	0.05	21	0.05
22	20 thru 23	380	0.15	20	0.10
26	24 thru 27	374	0.4	18	0.6
30	28 thru 31	345	1.0	19	1.0
34	32 thru 35	335	1.7	20	1.7
38	36 thru 39	328	2.6	20	2.3
42	40 thru 43	286	3.8	14	2.4
46	44 thru 47	278	4.5	16	4.1
50	48 thru 51	249	5.6	15	5.3
54	52 thru 55	105	6.2	6	6.0

Age (mos.) at eruption of first tooth in total sample

N	MEAN	S.D.	MINIMUM	MAXIMUM
354	7.3	1.7	1.7	11.5*

* Seven additional infants in the study were discharged at ages after 11½ months but "before first tooth erupted."

age 3 months, the prescribed daily dietary afforded 399 cc. evaporated milk, 1970–2050 International units of vitamin C, 150–250 Sherman-Chase units of vitamin B_1, and 0–810 U.S.P. units of vitamin D (less than 165 units for 31%, 250–600 units for 43% and 675–810 units for 26%). On the basis of "minute roentgen changes," 21 of the infants were diagnosed at different ages as having "mild" or "preclinical" scurvy—"9 of the 21 had some roentgen evidence of rickets at the time a diagnosis of mild scurvy was made" while "craniotabes or beading was recorded as a clinical finding in 17 of the 21 cases at some time before mild scurvy was evident" (6, p. 580). Eruption analyses were made for

both the scurvy subgroup and the total sample—the results are shown in Table VII. As may be confirmed from inspection of Table VII, selected findings for the total sample were: Earliest age of tooth eruption, 1.7 months; mean eruption in infants 7 months of age, 1 tooth; mean age at eruption of first tooth, 7.3 months; mean number of teeth erupted at ages 9 and 12 months, 3 and 6 respectively. Mean age for appearance of the first tooth in the mild scurvy subgroup was 8.1 months. The difference in eruption between the total sample and the mild scurvy subgroup was "not statistically significant."

Notations pertaining to "time of eruption of the teeth" were published by May and Wygant (10) on 48 "normal full-term" infants, 17 "weakling" infants, and 16 "premature" infants. "All of the infants came from the indigent class" (10, p. 356): Roughly two-thirds were White and one-third Negro. They were "enrolled as newborn infants in the Herman Kiefer Hospital, Detroit." "All were studied for a period of at least six months and many . . . for a year or more" (10, p. 358). Throughout the study, all subjects were "negative" for rickets, with the exception of 1 premature infant whose roentgenograms showed slight rachitic evidence around the age of 3 months. All were (a) fed irradiated evaporated milk assayed at 135 U.S.P. units of vitamin D per reconstructed quart and (b) "started on orange juice by the end of the second week" (10, p. 358). The percentages of each group lacking any eruption of teeth at ages 6 and 8 months are listed in Table VIII. For the "full-term" and "weakling" groups combined, there were 21 White males and 23 White females: At age 6 months, 33% of the males and 21% of the females showed eruption of 1 or more teeth. Individual variations among the "full-term" group included the following: A Negro female who erupted her first tooth at age 12 months, another who erupted her first tooth at 3½ months and had all 20 erupted at 12 months; a White female with 8 teeth erupted by 7 months, another who did not erupt her first tooth until 7½ months but had 12 erupted by 12 months; a White male who erupted 7 teeth in 1½ months (first tooth at 6 months, 7 at 7½ months), a Negro female who erupted 8 teeth in 2½ months (first tooth at 6½ months, 8 at 9 months), and a Negro male who erupted 8 teeth in 3 months (first tooth at 5½ months, 8 at 8½ months).

In 1942 Doering and Allen (4) published an analysis of eruption data for the deciduous teeth collected 1930–41 at the Center for Research in Child Health and Development, Harvard School of Public Health. The subjects were full-term "clinically normal" infants born at the Boston Lying-in Hospital. They were "of American or North European parentage"—roughly half of Irish descent. Information on occupation of the fathers yielded around 30% unskilled or semiskilled, 50% skilled, and 20% managerial or professional. Examinations were made "at birth, at three months' intervals up to one year, and at six months' intervals thereafter" (4, p. 113): "The limits of variation in the age at examination" were 7 days up to 2 years and 10 days at older ages. The examinations included comprehensive medical evaluations and afforded the parents both general and individualized "advice as to feeding and care" (4, p. 31). "A tooth was considered erupted if it had ruptured the membrane of the gum and was in sight"

(4, p. 113). Selected quantitative results are presented in Table IX. Utilizing values from this table together with the results from further analyses, Doering and Allen deduced the following findings:

1. "No tooth in any child had erupted at three months of age. . . all children examined at 15 months had at least one tooth" (4, pp. 113, 120). At intermediate ages, the percentages of children with no teeth erupted were 80 (6 months), 20 (9 months), and 2 (12 months).

2. Of the 223 children examined at 2½ years of age, 71% showed eruption of the full complement of deciduous teeth: of the 218 examined at 3 years of age, only 4 % "failed to have all their teeth" (4, p. 115).

3. "In general the deciduous teeth erupt in the following order: central incisors, lateral incisors, first molars, cuspids, and second molars" (4, p. 115). For incisor teeth, the lower centrals tended to precede the upper centrals, and the upper laterals the lower laterals.

4. "From the original records it was noticed that in 13 males and in 14 females out of approximately 110 males and 110 females, certain discrepancies in order of eruption occurred. Most frequently in this small group the lower lateral incisors erupted after the first molars and at times the cuspids appeared before the first molars. Occasionally all the mandibular teeth were present when there were no maxillary cuspids or second molars present" (4, p. 115).

TABLE VIII

Deciduous Dentition in Infants of Lower Classes Fed Irradiated Evaporated Milk

Data collected in Detroit on White and Negro infants

	AGE 6 MOS.		AGE 8 MOS.	
	N	Percentage lacking eruption	N	Percentage lacking eruption
Premature (birthwt. less than 5½ lbs.).....	16	100	10	80
Weakling (birthwt. 5½ to 6½ lbs.).....	17	70	10	50
Full-term (birthwt. 6½ to 10 lbs.).....	48	70	31	32

5. For 95% of the subjects, the period during which the deciduous dentition erupted varied between 18 months and 30 months. One subject (male) "erupted 20 teeth in less than 12 months before he was 18 months old" (4, p. 120); another subject (female) had an eruption period of almost 3 years.

6. The mean number of teeth erupted was slightly higher for males than for females at all ages from 6 to 30 months. Largest sex differences were obtained at 9 and 12 months.

7. Individual differences in number of teeth present in the mouth was greatest at 12 and 18 months. Also, more teeth were found to erupt "between the ages of 12 and 18 months. than at any other time" (4, p. 115).

8. "The tooth with the greatest variation in age of eruption is the second molar. One child may have this tooth at 18 months, but another may not have it at 3 years" (4, p. 117).

A second 1942 study on "eruption of deciduous teeth" was reported by Robinow, Richards and Anderson (17). The subjects were "presumably normal, healthy, White children" enrolled for seriatim study with the Samuel S. Fels Research Institute, Yellow Springs, Ohio. They were "from families of average or better than average economic status . . . about equally urban and rural" (17, p. 328). Examinations were made "every month up to 6 months, every

TABLE IX

Eruption of Deciduous Teeth in Boston, "Middle Class," Clinic Supervised Childr

Number of teeth erupted at different ages

AGE	N	MEAN	S.D.	MINIMUM	PERCENTILES			MAXIMUM
					25	50	75	
Males								
mos.								
6	127	0.4	0.9	0	0	0	0	4
9	131	3.4	2.3	0	2	4	6	8
12	131	6.0	1.9	0	5	6	8	11
18	118	12.5	3.0	5	11	12	16	20
24	113	16.8	2.2	11	16	16	19	20
30	110	19.2	1.4	16	19	20	20	20
36	107	19.9	0.6	16	20	20	20	20
Females								
6	143	0.3	0.8	0	0	0	0	5
9	132	2.8	2.4	0	1	2	4	8
12	137	5.6	2.3	0	4	6	7	12
18	123	12.4	2.7	4	12	12	14	16
24	112	16.6	2.2	12	16	16	18	20
30	113	19.1	1.6	12	18	20	20	20
36	111	19.9	0.5	16	20	20	20	20

Percentage of children with right and left teeth of each type

TEETH	AGE IN MONTHS						
	6	9	12	18	24	30	36
Males							
Lower first incisors	17	75	97	100			
Upper first incisors	2	52	90	100			
Upper second incisors		26	68	96	100		
Lower second incisors		5	28	92	99	100	
Upper first molars			1	80	100		
Lower first molars				78	100		
Upper canines			1	35	91	100	
Lower canines				31	89	100	
Lower second molars				3	28	82	97
Upper second molars				2	26	75	95
Females							
Lower first incisors	13	68	95	100			
Upper first incisors	1	40	83	100			
Upper second incisors		14	53	94	100		
Lower second incisors		8	26	93	100		
Upper first molars			3	82	100		
Lower first molars			2	81	100		
Upper canines				33	88	97	100
Lower canines				27	88	99	100
Lower second molars				1	28	78	99
Upper second molars				1	25	75	96

1½ months until 18 months, every 2 months until 3 years, every 3 months until 6 years. . . . In addition, the mothers were asked to record the date of eruption for each tooth" (17, p. 127). "Teeth were considered erupted as soon as part of the crown had pierced the gum" (17, p. 127). "Eruption ages for all 20 deciduous teeth were obtained on 64 children" (31 males, 33 females; 1,280 teeth), and eruption ages for another 700 deciduous teeth on an additional 175 children. "The figures for right and left homologous teeth were combined" (17, p. 127). Table X affords the numerical findings. Discussion of findings treated order of tooth eruption and sex differences in eruption:

TABLE X

Eruption of Deciduous Teeth in Ohio Children of Middle and Upper Classes

	MALES		FEMALES		BOTH SEXES
	Mean	S.D.	Mean	S.D.	Mean
Age at eruption (mos.) for pairs of homologous teeth					
Lower central incisors	7.3	1.6	7.8	2.1	7.6
Upper central incisors	9.1	1.5	9.6	2.0	9.4
Upper lateral incisors	10.4*	2.4	11.9	2.7	11.1
Lower lateral incisors	13.0	2.8	13.8	3.6	13.4
Upper first molars	16.0	2.3	15.7	2.3	15.8
Lower first molars	16.2	1.9	15.6	2.2	15.9
Upper canines	18.9	2.7	20.1	3.2	19.5
Lower canines	19.3	2.9	20.2	3.4	19.8
Lower second molars	25.9	3.8	27.1	4.2	26.5
Upper second molars	27.6	4.4	28.4	4.3	28.0
Number of teeth erupted at each of eight ages					
6 months	0.4	0.9	0.4	0.8	0.4
9 months	3.1	1.7	2.2	2.0	2.7
12 months	6.5	1.9	5.6	2.6	6.1
18 months	12.9	2.5	12.2	2.6	12.6
24 months	17.2	1.7	16.2	2.2	16.7
30 months	19.4	1.4	18.6	1.6	19.0
36 months	20.0	0.4	19.9	0.7	20.0
42 months			20.0	0.0	20.0

* 10.5 in another table.

1. "Boys were slightly advanced over girls in the eruption of all teeth except the first molars" (17, p. 133). The eruption of incisor and canine teeth was more variable in females than in males.

2. The characteristic sequence of eruption was incisors (lower first incisors, upper first incisors, upper second incisors, lower second incisors); first molars, canines; and second molars (lowers before uppers). The investigators stated: "Most of our children followed this pattern though the variations were quite common. Children, the majority of whose teeth erupted late, tended to show more variations from the above order than early eruptors. Changes in the order

were most frequently brought about by the variability of the lateral incisors" (17, pp. 129–130).

Meredith (12), in 1943, analyzed data for eruption of the deciduous teeth obtained at quarter-year ages from 3 months to 2 years. The data were gathered 1938–42 on 225 White infants—112 males and 113 females—enrolled for developmental examinations at the well-infant laboratory of the Iowa Child Welfare Research Station, University of Iowa. A tooth was recorded as erupted if any part of it had perforated the gingiva. Upwards of 90 per cent of the subjects

TABLE XI

Deciduous Dentition at Each of Eight Ages

(Data collected 1938–42 on Iowa City infants of middle and upper classes)

Means and extreme values for number of erupted teeth

AGE	MALES				FEMALES			
	N	Mean	Minimum	Maximum	N	Mean	Minimum	Maximum
mos.								
3	38	0.0	0	0	36	0.1	0	2
6	53	0.7	0	4	53	0.6	0	4
9	43	3.6	0	8	54	3.3	0	8
12	36	6.8	2	12	31	6.1	0	12
15	22	10.4	6	16	33	9.2	4	15
18	32	12.4	7	16	36	12.1	7	16
21	31	15.1	9	20	28	14.7	10	20
24	19	16.9	14	20	15	16.4	10	20

Percentage of subjects with different numbers of erupted teeth

	MALES					FEMALES				
	0	1–4	5–8	9–14	15–20	0	1–4	5–8	9–14	15–20
3	100					97	3			
6	64	36				68	32			
9	14	51	35			9	59	32		
12		8	86	6		3	23	68	6	
15			41	45	14		6	42	49	3
18			13	59	28			11	64	25
21				35	65				39	61
24				16	84				13	87

were of northwest European ancestry. About 25 per cent were examined at one age, 35 per cent at two ages, and 40 per cent at three or more ages. Approximately 50 per cent of the fathers were engaged in professional or managerial occupations and 50 per cent in skilled or semiskilled occupations. The findings from two types of analysis are displayed in Table XI.

Additional findings were:

1. The number of teeth erupting within a given period varied markedly for different individuals. The maximum eruptions found to occur within 3 selected periods were as

follows: Two subjects erupted 16 teeth within a period of 1 year (a female, no teeth at 6 months, 16 at 18 months; a male, no teeth at 9 months, 16 at 21 months); 3 other subjects erupted 14 teeth within a period of 9 months (a female, 6 teeth at 1 year, 20 at 21 months; 2 males, 2 teeth at 6 months, 16 at 15 months); another 2 subjects erupted 10 teeth within a period of 6 months (a female, 2 teeth at 6 months, 12 at 1 year; a male, no teeth at 9 months, 10 at 15 months).

2. The predominant eruption sequence was incisors—lower first incisors, upper first incisors, upper second incisors and lower second incisors; first molars; canines; and second molars. Exceptions to this sequence included eruptions of (a) upper first incisors before lower first incisors, (b) upper second incisors before upper first incisors, (c) lower second incisors before upper second incisors, (d) lower first molars before incisors—one female subject was born with the lower first molars erupted, (e) upper and lower first molars before upper second incisors, (f) upper and lower first molars before lower second incisors, (g) lower canines before second incisors or first molars, (h) upper canines before lower second incisors, (i) lower canines before upper or lower first molars, (j) upper and lower canines before upper first molars, and (k) upper and lower second molars before canines.

Deciduous eruption materials for a 1944 investigation by Sandler were amassed from "examination of 1,962 White children of fairly low economic status but under regular nutritional and medical supervision" (18, p. 146). The subjects resided in Brooklyn, New York, and were enrolled at 7 "well-baby clinics . . . in the Brownsville and Bushwick Health Districts" (18, p. 140). Data were obtained on all White children attending the clinics during the fall and winter of 1941–42. "The definition of an erupted tooth was limited to its appearance through the gums" (18, p. 140). Table XII brings together selected results from statistical reduction. ". . . no child under 4 months of age had a tooth erupted . . . all of the children over 34 months of age had all of their deciduous teeth erupted" (18, p. 141). The oldest age at which no teeth were found to have erupted was 13 months: the youngest age at which all 20 teeth were erupted was also 13 months. Four teeth were present in children as young as 5 months and as old as 17 months, 8 teeth in children aged 9 months and 24 months; 12 teeth in children 11 months and 25 months; 16 teeth in children 11 months and 32 months. Sandler reported finding no systematic tendency for the teeth of the right side to erupt earlier than those of the left, or vice versa.

Mean ages for eruption of the first incisor and of the full deciduous complement were reported in 1945 by Rhoads, Rapoport, Kennedy and Stokes (16). Collection of the data was carried out 1936–42 through the Out-Patient Department of the Children's Hospital of Philadelphia. The subjects were 233 full-term male children—134 White and 99 Negro—residing in families "of a low socio-economic level" and given dietary supervision from an average age of 6 postnatal weeks. Children were "classed as Negro where they had any known trace of Negro blood" (16, p. 437): The White subjects were "in large proportion of northern European stock, with approximately one-fourth of Italian descent" (16, p. 416). All of the subjects were followed to 2 years of age, 208 to 3 years, and 153 to 4 years. Examinations were made once a month during the first year and every 2 months thereafter. From the time of the first examination all subjects were prescribed orange or tomato juice and an evaporated milk formula with added carbohydrate. Four dietary subgroups were formed—

TABLE XII

Eruption of Deciduous Teeth in New York Children of Lower Classes

Order and age (mos.) of eruption of teeth

	EARLIEST AGE ERUPTION OBSERVED	PERCENTILES			LATEST AGE ERUPTION LACKING
		25	50	75	
Lower first incisor	4	6.1	7.8	9.4	17
Upper first incisor	5	8.1	9.6	11.0	15
Upper second incisor	6	9.9	11.5	13.0	21
Lower second incisor	6	10.5	12.4	14.3	27
Upper first molar	8	13.4	15.1	16.8	27
Lower first molar	8	13.8	15.7	17.6	27
Lower canine	8	16.2	18.2	20.0	29
Upper canine	8	16.3	18.3	20.2	29
Lower second molar	8	23.5	26.0	28.3	34
Upper second molar	8	23.7	26.2	28.5	34

Number of teeth erupted at different ages

AGE GROUP		N	MINIMUM	PERCENTILES			MAXIMUM	MEANS		
Midpoint	Range			25	50	75		Male	Female	Both sexes
mos.	*mos.*									
Under 4 months		330	0	0	0	0	0			
5	4.5– 5.5	86	0	0	0	0	4	0.4	0.2	0.3
6	5.5– 6.5	95	0	0	0	2	6	0.8	0.6	0.7
7	6.5– 7.5	77	0	0	0	2	6	1.4	0.8	1.2
9	8.5– 9.5	73	0	1	2	4	9	3.2	2.8	3.0
12	11.5–12.5	56	0	4	6	8	17	6.6	5.8	6.2
15	14.5–15.5	66	3	7	9	12	16	10.2	8.2	9.2
18	16.5–19.5	124	4	12	13	16	18	12.8	13.2	13.1
21	19.5–22.5	119	4	14	16	16	20	15.6	15.4	15.5
24	22.5–25.5	110	8	16	16	18	20	16.5	16.7	16.6
30	28.5–31.5	70	12	18	20	20	20	19.0	19.0	19.0
Over 34 months		110	20	20	20	20	20	20.0	20.0	20.0

TABLE XIII

Mean Age (mos.) at Eruption of First Incisor and Full Deciduous Complement

Data for Philadelphia males of "low income families" on four prescribed diets

GROUP	WHITE MALES				NEGRO MALES			
	First incisor		20 teeth		First incisor		20 teeth	
	N	Mean	N	Mean	N	Mean	N	Mean
I	48	7.5	45	28.0	32	7.8	30	28.7
II	24	8.0	24	27.2	17	8.2	17	28.1
III	20	8.3	18	28.9	19	8.5	19	27.8
IV	42	8.0	41	28.8	31	8.7	31	28.4
Groups I–IV	134	7.9	128	28.2	99	8.3	97	28.3

the objective in this was to determine the influence of supplements of vitamins D, A, and B complex on the deciduous dentition of children fed evaporated milk as their sole milk supply. Groups I, II, and III received "110 U.S.P. units of vitamin D daily in the form of irradiated evaporated milk"; Groups II and III also were given approximately 2,250 U.S.P. units of vitamin A daily in 10 drops of carotene; and Group III received roughly 250 units of vitamin B_1 and 250 Sherman units of vitamin B_2 daily through the medium of brewer's yeast powder. Group IV was fed "non-irradiated evaporated milk plus 3 teaspoons of cod-liver oil daily" (16, p. 418). For all groups, visits were made to the homes every 2 weeks "in order to deliver the evaporated milk and vitamin supplements, and to keep in touch with the home care of the child" (16, p. 417). At 6 months of age the common dietary included "banana, cereal, and pureed vegetables and fruits, and at 1 year, potato, meat and egg" (16, p. 417). "Incidence of infection . . . was approximately the same for all groups" (16, p. 419). The dental examinations were made by Drs. Norman Weiselberg and Albert Cohen. Table XIII itemizes the findings.

It was concluded:

1. "Whether the child was fed 110 units of vitamin D in irradiated evaporated milk or 1,500 units in cod-liver oil apparently made no difference in the age at eruption of teeth, nor did supplementation with carotene or yeast" (16, p. 447).

2. ". . . there was no striking difference in the eruption times of the White and Negro children" (16, p. 448).

Stearns and others (21) studied deciduous tooth eruption in White infants especially housed for research on mineral and vitamin metabolism at the Children's Hospital, University of Iowa. "The date of the appearance of each tooth was a part of the daily records kept for each infant by the ward nurses. The day on which any of the incisal or occlusal portion of a tooth appeared through the gum was recorded as the date of eruption" (21, p. 506). Over the period 1930–45 dental records were secured on 43 males and 18 females under continuous observation from early postnatal life to at least 9 months of age. The infants were fed controlled diets built around a whole or evaporated milk mixture containing 6% added carbohydrate, and usually acidified with lactic or citric acid. One ounce of orange juice daily was given to infants under 4 months and 2 ounces to older infants. Egg yolk or iron citrate was added to the daily diet at 3 to 4 months, sieved vegetables at 5 months and sieved fruits at 6 months. The daily intake of vitamin D varied from less than 100 U.S.P. units for some infants to upwards of 4,000 U.S.P. units for others. Analyses were made on the total group of males and females and on 2 dietary-health subgroups. One subgroup consisted of 14 males and 8 females considered to have "superior" dietary and health protocols: Each infant ingested 300 to 400 U.S.P. units of vitamin D daily and was free from both severe acute and mild chronic illnesses. The remaining 29 males and 10 females constituted the other ("poorer") subgroup. In the first 4 sections of Table XIV are findings obtained utilizing the total sample, and in the last section findings for each of the 2 dietary-health subgroups. A few of the results which may be drawn from this table are: (1) tooth eruption

TABLE XIV

Eruption of Deciduous Teeth in White Infants on Controlled Dietary Intakes

Number of teeth at ages 6 and 9 months

SEX	AGE	N	MEAN	PERCENTAGE OF INFANTS WITH:				
				0	1–2	3–4	5–6	7–8
	mos.							
Females	6	18	0.2	89	11			
Males	6	43	0.8	60	33	5	2	
Females	9	18	2.9	11	56	11	16	6
Males	9	43	3.8	12	23	30	21	14

Age (mos.) at eruption of first tooth

SEX	N	MINIMUM	PERCENTILES:			MAXIMUM
			25	50	75	
Females	18	5.0*	6.4	7.3	7.8	10.0+†
Males	43	4.0	5.4	6.7	8.0	11.2‡

Individual differences in the first, third and sixth teeth erupted

PERCENTAGE OF INFANTS OF BOTH SEXES ERUPTING:	ORDER OF ERUPTION		
	First tooth	Sixth tooth	Third tooth
Lower first incisor	96	2	3
Upper first incisor	2	72	25
Upper second incisor	2	20	53
Lower second incisor		6	19
Number of subjects	58	49	36

Individual differences in order of eruption of lower central incisors

	RIGHT TOOTH FIRST BY:		BOTH TEETH TOGETHER (LESS THAN 1 WEEK APART)	LEFT TOOTH FIRST BY:	
	4 to 6 wks.	1 to 3 wks.		1 to 3 wks.	4 to 8 wks.
Percentage	12	18	45	21	4

Eruption of teeth in 2 dietary-health subgroups

GROUP	NUMBER		MEDIAN ERUPTION AGE FOR FIRST TOOTH		MEAN NUMBER OF TEETH AT AGE 9 MONTHS	
	Males	Females	Males	Females	Males	Females
			mos.	*mos.*		
"Superior"	14	8	5.6	7.3	4.7	2.9
"Poorer"	29	10	7.1	7.4	3.4	2.9

* The youngest age of tooth eruption for an infant in this research laboratory is 2.4 months. The subject was a female available for study until 6 months of age.

† Two females left the study before erupting any teeth—one at 9 months and the other at 10 months.

‡ For 1 male, it is not known at what age after 9 months the first tooth pierced the gum.

tended to occur earlier in males than in females; (2) number of erupted teeth at age 9 months varied from 0 to 8 for infants of each sex; (3) there was no systematic tendency for the lower left first incisor to erupt earlier than the lower right, or *vice versa*; (4) the sixth tooth to erupt was an upper second incisor in 53% of the infants, an upper first incisor in 25%, and a lower second incisor in 19%; (5) male infants classified as constituting a "superior" dietary-health subgroup showed unusually early tooth eruption.

GENERALIZATIONS

The foregoing investigations on eruption of the deciduous dentition in North American children yield the following generalizations:

1. Rarely do infants erupt teeth before the end of their fourth postnatal month. Reported instances of tooth eruption at younger ages include a White female born with 2 teeth (12), a White or Negro infant erupting a tooth at 1.7 months (6), 3 White and/or Negro infants erupting their first teeth at 2 months (1), a White female erupting a tooth at 2.4 months (21), an American Indian female with 2 teeth erupted at 2.5 months (8), a White infant of each sex showing eruption of a tooth at 3 months (22), and a Negro female erupting a tooth at 3.5 months (10). Aggregating the relevant samples of White infants studied (5, 18, 21, 2, 4, 7, 10, 12, 17, 22), it is estimated that the frequency of tooth eruption prior to 4 months of age is less than 1%.

2. The mean age for eruption of the first tooth is $7\frac{1}{2}$ months. This is the exact composite mean derived by pooling the data on 1,095 infants—about 70% White and 30 per cent Negro—available from 5 investigations (1, 6, 16, 21, 22). The most diverse averages reported for eruption of the first tooth are 5.6 months and 8.7 months. The latter was obtained on 31 Negro males prescribed a diet of "evaporated milk plus 3 teaspoons of cod-liver oil daily"; the former on 14 White males living under controlled conditions, ingesting 300–400 U.S.P. units of vitamin D daily, and affording health protocols "negative" for severe acute and mild chronic illnesses.

3. A few infants do not begin to erupt teeth until early in the second postnatal year. This may be confirmed by reference to the records of first eruption at 13 months (1, 18), during the first quarter of the second year (4, 12), and at 15 and 16 months (22). Combining the pertinent materials accumulated in 8 studies (1, 4, 5, 6, 10, 12, 18, 22), it appears that approximately 1% of White infants erupt their first tooth after 1 year of age. Incidentally, after 2 months allowance is made for the amount of prematurity, all of the Dionne quintuplets are found to have erupted teeth by the end of the first year (3).

4. At 6 months of age, approximately 1 infant in 3 is characterized by 1 or more erupted teeth. In a series of 576 White infants, roughly two-thirds have been found to lack tooth eruption and one-third to exhibit from 1 to 6 erupted teeth (4, 10, 12, 18, 21). Obviously, with a distribution of this type the mean and median do not coincide. The median is zero, *i.e.*, the typical infant age 6 months has no erupted teeth. The mean, as derived from records for number of erupted teeth on more than 1,100 White and Negro infants (4, 5, 6, 12, 17, 18, 21), is 0.4.

5. At age 9 months, the average infant has 3 erupted teeth. Three is the median for 263 "middle class" White infants given pediatric supervision (4) and the mean for 670 White infants of varying socio-economic status and health care (4, 5, 12, 17, 18, 19, 21). The usual range of individual variation is from 0 to 8 teeth (4, 5, 12, 21): One investigator reports a maximum of 9 teeth (18). About 10% of White infants have no teeth, 80% between 1 and 6 teeth, and 10% more than 6 teeth (4, 12, 18, 21). For each sex separately, the means are 3.4 erupted teeth in 350 males and 2.7 in 320 females (4, 5, 12, 17, 18, 19, 21): Among those infants erupting over 6 teeth by 9 months, there are roughly 3 times as many males as females (12, 21). The extreme means reported on males are 2.6 for 14 "Sicilian infants from the lower East Side of New York" (19) and 4.7 for 14 infants living under controlled conditions and having "superior" histories for nutrition and health (21). The female means are more homogeneous.

6. In infants 1 year of age, the number of erupted teeth is rarely less than 2 or more than 10. A few infants have no teeth (4, 12, 18) and a few 12 teeth (4, 5, 12) or more—examples of more than 12 erupted teeth are a White infant with 17 (18) and a Negro infant with 20 (10). Fifty per cent of 1-year-old infants have between 4 and 8 teeth (4, 18). Composite means are 6.0 for 700 infants of both sexes (approximately 90% White and 10% Negro), 6.3 for 260 White males, and 5.7 for 270 White females (4, 5, 6, 12, 17, 18).

7. The typical, or median, number of erupted teeth is 12 in children 18 months of age and 16 in children 2 years of age. For White infants age 18 months, about 10% have 4 to 8 teeth, 85% 9 to 16 teeth, and 5% 17 to 20 teeth (4, 12, 17, 18). Composite mean values at this age are 12.7 for 245 males and 12.5 for 255 females (4, 12, 17, 18). White infants age 2 years are dispersed with roughtly 15% having 8 to 14 teeth, 60% 15 to 18 teeth, and 25% 19 or 20 teeth (4, 12, 18, 22). Composite means at 2 years are 16.9 for 260 males and 16.5 for 250 females (4, 12, 15, 17, 18, 22).

8. At 2½ years of age, 30% of White children have 12 to 19 erupted teeth and 70% the full deciduous complement. The mean number of teeth at this age is 19.2 for males and 19.0 for females (4, 17, 18): The typical (median) child has 20 teeth (4, 18). Data for exact age at eruption of the last deciduous tooth have yielded means of 28.2 months on 128 White males and 28.3 months on 97 Negro males (16). Roughly 90% of children complete eruption of the deciduous dentition between 2 and 3 years of age. Instances of completion earlier than 2 years include a Negro child with 20 teeth at 12 months (10), a White child with 20 teeth at 13 months (18), a White child with 20 teeth at 18 months (4), 2 American Indian children with 20 teeth at 20 months (8), and White children with 20 teeth at 21 months (12, 18). Individuals still lacking eruption of the full deciduous complement have been observed at 36 months (4, 17) and 37 months (15, 19). When deductions of 2 months are made to correct for the amount of prematurity, 4 of the Dionne quintuplets are found to have erupted the full deciduous complement by 36 months and 1 (Yvonne) after 38 months (3).

9. There are wide individual differences in the timing of eruption of deciduous teeth: Some children erupt a given tooth early, others late; some erupt the full deciduous complement in a short period, others spread their 20 eruptions over an

extended time interval. From the standpoint of number of teeth present in the mouth at different ages, variation is greatest during the first half of the second year. Records manifesting the maximum scatter attainable (*i.e.*, no erupted teeth in one child and a full complement in another) have been obtained on Negro subjects age 12 months (10) and on White subjects age 13 months (18). The standard deviation is largest around 18 months (4, 17): Minimum and maximum records for number of erupted teeth at this age are 4 and 20 (4). Turning to consideration of the age span during which given numbers of teeth are found, 4 teeth have been reported in children as young as 5 months and as old as 20 months, 8 teeth in children 7 and 24 months, 12 teeth in children 11 and 30 months, and 16 teeth in children 11 and 37 months (10, 11, 18, 19). The duration of the deciduous eruption period varies from less than 1 year to almost 3 years (4, 10): For over 90% of children the span of time within which the first dentition erupts is between 18 and 30 months (4). Instances are on record of children erupting 7 teeth in 1.5 months, 12 teeth in 6 months, and 20 teeth in 9 months (10, 12).

10. The usual order of eruption for the deciduous teeth is incisors, first molars, canines, second molars. In greater detail, the characteristic eruption sequence has been established as lower first incisors, upper first incisors, upper second incisors, lower second incisors, first molars (upper and lower), canines (upper and lower), lower second molars, upper second molars (2, 4, 17). It follows that the 6 erupted teeth of the typical child age 1 year are the lower first incisors and the upper first and second incisors. Similarly, the 16 erupted teeth of the typical child age 2 years are the first and second incisors, the first molars, and the canines. For about 95% of individuals the first tooth to erupt is a lower first incisor (21): Occasionally eruption begins with an upper first incisor, upper second incisor, or first molar (3, 12, 21). The third tooth to erupt, while ordinarily an upper first incisor, is an upper second incisor in approximately 20% of infants and a lower second incisor in roughly 5% (21). Additional exceptions to the predominant pattern include the eruption of first molars before second incisors, canines before second incisors, canines before first molars, and second molars before canines (3, 4, 12, 17). Obviously, when these exceptions are elaborated for specific teeth or tooth pairs an extensive list of variations accrues (3, 4, 12). One study reports: "Occasionally all the mandibular teeth were present when there were no maxillary cuspids or second molars" (4). Another, after noting that variations in the sequence of eruption are "common," states: "Children, the majority of whose teeth erupted late, tended to show more variations . . . than early eruptors" (17).

11. *Sex:* At all ages from 9 months to 2 years teeth tend to erupt earlier in males than in females.[3] *Race:* While materials on Negro subjects are limited, it appears there is "no striking difference" in average age of tooth eruption for

[3] This finding stands in sharp contrast with certain statements in a recent book on sex differences. To quote: "It is entirely natural for the little girl to be ahead. . . in teething." And again, ". . . the girl usually leads in every phase of tooth development" (Scheinfeld, Amram, *Women and men*, 1944, pp. 41, 47–48).

White and Negro children (10, 14, 16). The few observations on American Indian children apparently conform to the same general eruption sequence as followed by White children, but suggest an earlier timing of eruption. *Diet and disease:* No association has been found between the age of eruption of deciduous teeth and early addition to the diet of vitamin B through the medium of brewer's yeast powder or a water-soluble extract of rice polishings (14, 16). One investigation obtained "no difference in the age at eruption of teeth" between 150 children fed 110 U.S.P. units of vitamin D daily in irradiated evaporated milk and 70 children fed 1500 U.S.P. units daily in cod-liver oil (16). In another study it was found that a group of 14 male infants ingesting 300–400 U.S.P. units of vitamin D daily, and also having superior records for freedom from illness, erupted their incisor teeth at ages earlier than those reported on any other group (21).[4] The age of tooth eruption in a small group of infants developing mild scurvy and mild (subclinical) rickets was found not to differ from that for healthy infants (6). *Prematurity:* There are a limited number of observations tending to support the hypothesis that tooth eruption during the first postnatal year occurs later in infants born prematurely than in infants born "at term" (3, 10).

SUMMARY

This paper treats the order and age of eruption of the deciduous teeth in North American children. Its purpose is twofold: To assemble the available research materials on the problem and to present a synthesis of these materials. The findings from 22 investigations (including 2 not previously reported) are reviewed. These findings are then ordered and integrated in a series of statements which epitomize present knowledge on the problem.

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[4] Without minimizing the need for further study of the relationship between intake of vitamin D and age of tooth eruption, it is pertinent to mention the present hypothesis of Drs. Philip C. Jeans and Genevieve Stearns. For many years of controlled research covering the first year of postnatal life, these authors have formulated the following view: a. 300–800 U.S.P. units of vitamin D daily promotes accelerated rate of growth in stature, increased calcium and phosphorus retentions, and early eruption of teeth; b. less than 125 units is ordinarily insufficient for "maximum effect"; and c. 1500–2000 units usually has a "depressing (toxic) action." In terms of this view there is no lack of harmony between the finding of early tooth eruption for infants receiving 300–400 units (maximum effect) and the findings of later ages of eruption both for infants fed 110 units (increasing effect but not maximum) and for those fed 1500 units (decreasing effect).

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